Residual Risk Assessment for Eight Source Categories:

Polysulfide Rubber Production
Ethylene Propylene Rubber Production
Butyl Rubber Production
Neoprene Production
Epoxy Resins Production
Non-nylon Polyamides Production
Hydrogen Fluoride Production
Acetal Resins Production

by
EPA's Office of Air Quality Planning and Standards
Office of Air and Radiation
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Appendices

1 Introduction

Section 112 of the Clean Air Act (CAA) establishes a two-stage regulatory process for addressing emissions of hazardous air pollutants (HAPs) from stationary sources. In the first stage, section 112(d) (2) requires the Environmental Protection Agency (EPA, or the Agency) to develop technology-based standards for categories of industrial sources (e.g., petroleum refineries, pulp and paper mills, etc.) [1]. EPA has largely completed the initial Maximum Achievable Control Technology (MACT) standards as required under this provision. Under section 112(d)(6), EPA must review each of these technology-based standards at least every eight years and revise a standard, as necessary, "taking into account developments in practices, processes and control technologies." In the second stage, EPA is required under section 112(f)(2) to assess the health and environmental risks that remain after sources come into compliance with MACT. If additional risk reductions are necessary to protect public health with an ample margin of safety or to prevent adverse environmental effects, EPA must develop standards to address these remaining risks. This second stage of the regulatory process is known as the residual risk stage. For each source category for which EPA issued MACT standards, the residual risk stage must be completed within eight years of promulgation of the initial technology-based standard.

In December of 2006 we consulted with a panel from the EPA's Science Advisory Board (SAB) on the "Risk and Technology (RTR) Review Assessment Plan" and in June of 2007, we received a letter with the results of that consultation. We have incorporated suggestions from their key messages, where appropriate and relevant, in the risk assessments performed for the eight source categories. Further peer review of the RTR approach is planned to continue ensuring improvements in the risk assessment methodologies used.

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This document contains the methods and the results of risk assessments performed for eight source categories. The eight source categories are:

Polysulfide Rubber Production
Ethylene Propylene Rubber Production
Butyl Rubber Production
Neoprene Production
Epoxy Resins Production
Non-nylon Polyamides Production
Hydrogen Fluoride Production
Acetal Resins Production

The methods discussion includes descriptions of the methods used to develop refined estimates of chronic inhalation exposures and human health risks for both cancer and noncancer endpoints, as well as descriptions of the methods used to screen for acute health risks, chronic non-inhalation health risks, and adverse environmental effects. Since none of the screening assessments indicated any significant potential for chronic non-inhalation health effects, or environmental impacts including effects to threatened and endangered species, no further refinement of these assessments was performed. Screening assessments did not indicate a concern for acute health effects for five of the source categories. A more refined analysis was performed for two of the source categories for acute exposure impacts.

2 Methods

2.1 Emissions and source data

The 2002 National Emissions Inventory (NEI) Final Version 1 (made publicly available February 2006) served as the starting point for this assessment. Using the process MACT code[1], we developed a subset of this inventory that contains emissions and facility data for each of the eight source categories addressed. Next, we performed an engineering review of these using EPA engineers who were directly involved in the development of the MACT standards for these eight source categories, and/or who have extensive knowledge of the characteristics of these industries. NEI data was updated with industry supplied data as available. The goal of the engineering review was to identify readily-apparent limitations and issues with the emissions data (particularly those that would greatly influence risk estimates) and to make changes to the data set where possible to address these issues and decrease the uncertainty of the assessment. The emissions data and modifications made to the NEI data are discussed in the memo "Emissions Data Used in Residual Risk Modeling for the Eight Phase I RTR Source Categories" (see Appendix 1). Additionally, individual facilities were contacted to modify site specific data as a result of screening assessment results.

2.2 Dispersion modeling for inhalation exposure assessment

Both long- and short-term inhalation exposure concentrations and associated health risk from each facility in each of the source category of interest were estimated using the Human Exposure Model in combination with the American Meteorological Society/EPA Regulatory Model dispersion modeling system (HEM-AERMOD). The approach used in applying this modeling system is outlined below, and further details are provided in Appendix 2. HEM-AERMOD performs three main operations: atmospheric dispersion modeling, estimation of individual human exposures and health risks, and estimation of population risks. This section focuses on the dispersion modeling component. The exposure and risk characterization components are discussed throughout sections 2 and 3.

The dispersion model in the HEM-AERMOD system, AERMOD version 07026, is a state-of-the-science Gaussian plume dispersion model that is preferred by EPA for modeling point, area, and volume sources of continuous air emissions from facility applications [*2*]. Further details on AERMOD can be found in the AERMOD Users Guide [*3*]. The model is used to develop annual average ambient concentration through the simulation of hour-by-hour dispersion from the emission sources into the surrounding atmosphere. Hourly emission rates used for this simulation are generated by evenly dividing the total annual emission rate from the inventory into the 8,760 hours of the year.

[1] The tagging of data with MACT codes allows EPA to determine reductions attributable to the MACT program. The NEI associates MACT codes corresponding to MACT source categories with stationary major and area source data. MACT codes may be assigned either at the process level or at the site level in the point source data (e.g., the MACT code for municipal waste combustors (MWCs) is assigned at the site level whereas the MACT code for petroleum refinery catalytic cracking is assigned at the process level).

The first step in the application of the HEM-AERMOD modeling system is to predict ambient concentrations at locations of interest. The AERMOD model options employed are summarized in Table 2.2-1 and are discussed further below.

Table 2.2-1 AERMOD version 07026 model options for RTR II modeling

Modeling Option	*Selected Parameter for chronic exposure*
Type of calculations	Hourly Ambient Concentration
Source type	Point, area represented as pseudo point source
Receptor orientation	Polar (10 rings at 10-deg) Discrete (census block centroids)
Terrain characterization	Actual from USGS 1-degree DEM data
Building downwash	Not Included
Plume deposition/depletion	Not Included
Urban source option	No
Meteorology	1 year representative NWS from nearest site (122 stations)

Meteorological data for HEM-AERMOD is selected from a list of 158 National Weather Service (NWS) surface observation stations across the continental United States, Alaska, Hawaii, and Puerto Rico. In most cases the nearest station is selected as representative of the conditions at the subject facility. Ideally, when considering off-site meteorological data most site specific dispersion modeling efforts will employ up to five years of data to capture variability in weather patterns from year to year. However, because of limited availability of meteorological datasets in the needed modeling format, it was not practical to model five years of data and only a single year was modeled. While the selection of a single year may result in under-prediction of long-term ambient levels at some locations, likewise it may result in over-prediction at others. For each facility identified by its characteristic latitude and longitude coordinates, the closest meteorological station was used in the dispersion modeling. The average distance between a modeled facility and the applicable meteorological station was 40 miles (72 km). A sensitivity analysis performed for the 1996 National Air Toxics Assessment (NATA) examined the chronic inhalation risk variability attributable to the selection of the meteorology dataset location [7]. The analysis found that predicted risk estimates were up to 17% below and 84% above the risk estimates predicted by utilizing the closest station. Appendix 3 (Meteorological Data Processing Using AERMET for HEM-AERMOD) provides a complete listing of stations and assumptions along with further details used in processing the data through AERMET.

The HEM-AERMOD system estimates ambient concentrations at the geographic centroids of census blocks (using the 2000 Census), and at other receptor locations that can be specified by the user. The model accounts for the effects of multiple facilities when estimating concentration impacts at each block centroid. In this assessment, we combined only the impacts of facilities within the same source category, and assessed chronic exposure and risk only for census blocks with at least one resident (i.e., locations where people may reasonably be assumed to reside rather than receptor points at the fenceline of a facility). Chronic ambient concentrations were calculated as the annual average of all estimated short-term (one-hour) concentrations at each block centroid. Possible future residential use of currently uninhabited areas was not considered. Census blocks, the finest resolution available in the census data, are typically comprised of approximately 40 people or about ten households.

In contrast to the development of ambient concentrations for evaluating long-term exposures, which was performed only for occupied census blocks, worst-case short-term (one-hour) concentrations were estimated both at the census block centroids and at points nearer the facility that represent locations where people may be present for short periods, but generally no nearer than 100 meters from the center of the facility (note that for large facilities, this 100-meter ring could still contain locations inside the facility property). Since short-term emission rates were needed to screen for the potential for hazard via acute exposures, and since the NEI contains only annual emission totals, we applied the general assumption to all source categories that the maximum one-hour emission rate from any source was ten times the average annual hourly emission rate for that source. Average hourly emissions rate is defined as the total emissions for a year divided by the total number of operating hours in the year. This choice of a factor of ten for screening is was originally based on engineering judgment. To develop a more robust peak-to-mean emissions factor, and in response to one of the key messages from the SAB consultation on our RTR Assessment Plan, we recently performed an analysis using a short-term emissions dataset from a number of sources located in Texas (originally reported on by Allen *et al.* (2004)[*4*]). In that report, the Texas Environmental Research Consortium Project compared hourly and annual emissions data for volatile organic compounds for all facilities in a heavily-industrialized 4-county area (Harris, Galveston, Chambers, and Brazoria Counties, TX) over an eleven-month time period in 2001. We obtained the dataset and performed our own analysis, focusing that analysis on sources which reported emitting high quantities of HAP over short periods of time (see Appendix 4, Analysis of data on short-term emission rates relative to long-term emission rates). Most peak emission events were less than twice the annual average, and the highest was a factor of 11 times the annual average. The factor of ten is intended to cover both routinely-variable emissions and startup, shutdown, and malfunction (SSM) emissions. While there are some documented emission excursions above this level, our analysis of the data from the Texas Environmental Research Consortium suggests that this factor should cover more than 99% of the short-term peak gaseous or volatile emissions from typical industrial sources.

Census block elevations for HEM-AERMOD modeling were determined nationally from the US Geological Service 1-degree digital elevation model (DEM) data files, which have a spatial resolution of about 90 meters. Polar grid elevations (used in estimating short- and long-term ambient concentrations) were evaluated at the highest elevation of any census block in that sector. If a sector does not contain any blocks, the model defaults to the elevation of the nearest block. If the elevation is not provided for the emission source, the model takes the average elevation of all sectors of the nearest model ring.

In addition to utilizing receptor elevation to determine plume height, AERMOD adjusts the plume's flow if nearby elevated hills are expected to influence the wind patterns. For details on how hill heights were estimated and used in the AERMOD modeling see Appendix 2.

2.3 Estimating human inhalation exposure

For this assessment, we used the annual average ambient air concentration of each HAP at each census block centroid as a surrogate for the lifetime inhalation exposure concentration of all the people who reside in the census block. That is, this risk- analysis did not consider either the short-term or long-term behavior (mobility) of the exposed populations and its potential influence on their exposure.

We did not address short-term human activity in this assessment for two reasons. First, our experience with the 1996 and 1999 NATA assessments (which modeled daily activity using EPA's HAPEM model) suggests that, given our current understanding of microenvironment concentrations and daily activities, modeling short-term activity would, on average, reduce risk estimates about 25% for particulate HAPs; it will also reduce risk estimates for gaseous HAPs, but typically by much less. Second, basing exposure estimates on average ambient concentrations at census block centroids may underestimate or overestimate actual exposure concentrations at some residences. Further reducing exposure estimates for the most highly-exposed residents by modeling their short-term behavior could add a systematic low bias to these results.

We did not address long-term migration in this assessment nor population growth or decrease over 70 years, instead basing the assessment on the assumption that each person's predicted exposure is constant over the course of their lifetime which is assumed to be 70 years. In assessing cancer risk, 3 metrics are generally estimated, the maximum individual risk (MIR) which is defined as the risk associated with a lifetime of exposure at the highest concentration, the population risk distribution, and the cancer incidence. This assumption of not considering short or long-term population mobility does not bias the estimate of the theoretical MIR nor does it affect the estimate of cancer incidence since the total population number remains the same. It does, however, affect the shape of the distribution of individual risks across the affected population, shifting it toward higher estimated individual risks at the upper end and reducing the number of people estimated to be at lower risks, thereby biasing the risk estimates high.

When screening for potentially significant acute exposures, we used an estimate of the highest hourly ambient concentration at any off-site location as the surrogate for the maximum potential acute exposure concentration for any individual.

2.4 Multipathway and environmental risk screening

The potential for significant human health risks due to exposures via routes other than inhalation (i.e., multipathway exposures) were screened by first determining whether any sources emitted any pollutants known to be persistent and bioaccumulative in the environment (PB-HAP). There are 14 PB-HAP compounds or compound classes identified for this screening in EPA's Air Toxics Risk Assessment Library [*5*]. They are cadmium compounds, chlordane, chlorinated dibenzodioxins and furans, DDE, heptachlor, hexachlorobenzene, hexachlorocyclohexane, lead compounds, mercury compounds, methoxychlor, polychlorinated biphenyls, polycyclic organic matter, toxaphene, and trifluralin. PB-HAP emissions were not identified from any of the eight source categories, indicating that exposures due to non-inhalation routes of exposure were not significant. The lack of PB-HAP emissions from any of these source categories indicates a very low potential for adverse environmental effects due to exposures resulting from atmospheric deposition of PB-HAPs. Thus, even if sensitive species are present in the areas surrounding these facilities with no PB-HAP emissions, it is still reasonable to assume that there are no adverse effects to populations in the areas surrounding these facilities.

Additionally, we evaluated the potential for significant ecological exposures to non PB-HAP from exceedances of chronic human health inhalation thresholds in the ambient air near these facilities. Human health dose-response threshold values are generally derived from studies conducted on laboratory animals (such as rodents) and developed with the inclusions of uncertainty factors of that could be as high as 3000. Thus, these human threshold values are often significantly lower than the level expected to cause an adverse effect in an exposed rodent. It should be noted that there is a scarcity of data on the direct atmospheric impact of these HAPs on other receptors, such as plants, birds, and wildlife. Thus, if the maximum inhalation hazard in an ecosystem is below the level of concern for humans, we have concluded that mammalian receptors should be at no risk of adverse effects due to inhalation exposures from non PB-HAP, and have assumed, although some uncertainty exists, that other ecological receptors, such as plants, are similarly not at any significant risk from direct exposure to the emissions from these facilities.

2.5 Acute Risk Screening and Refined Assessments

In establishing a scientifically-defensible approach for the assessment of potential health risks due to acute exposures to HAP, we have followed the same general approach that has been used for developing chronic health risk assessments under the residual risk program. That is, we developed a tiered, iterative approach. This tiered, iterative approach to riskwas endorsed by the National Academy of Sciences in its 1993 publication “Science and Judgment in Risk Assessment” and subsequently was endorsed in the EPA’s “Residual Risk Report to Congress” in 1999.

The assessment methodology is designed to eliminate from further consideration those facilities for which we have confidence that no acute adverse health effects of concern will occur. To do so, we use what is called a tiered, iterative approach to the assessment. This means that we begin with a screening assessment, which relies on readily-available data and uses conservative (worst-case) assumptions. The result of this screening process is that either the facility being assessed poses no acute health risks (i.e., it “screens out”), or that it requires further refined assessment. A refined assessment could utilize site-specific data on the temporal pattern of emissions, the layout of emission points at the facility, the boundaries of the facility, and the local meteorology. In some cases, all of these site-specific data would be needed to refine the assessment; in others, lesser amounts of site-specific data could be used to determine that acute exposures are not a concern, and additional data collection would not be necessary.

With limited information on peak hourly emission rates, acute health risk screening was performed as the first step. We utilized conservative assumptions for emission rates, meteorology, and exposure location. We used the following worst-case assumptions in our screening approach:

- Peak 1-hour emissions are assumed to equal 10 times the average 1-hour emission rates.
- For facilities with multiple emission points, peak 1-hour emissions were assumed to occur at all emission points at the same time.
- For facilities with multiple emission points, 1-hour concentrations at each receptor were assumed to be the sum of the maximum concentrations due to each emission point, regardless of whether those maximum concentrations occurred during the same hour.
- Worst-case meteorology (from one year of local meteorology) is assumed to occur at the same time the peak emission rates occur. The recommended EPA local-scale dispersion model, AERMOD, is used for simulating atmospheric dispersion.
- A person is located downwind at the point of maximum impact during this same 1-hour period, but no nearer to the source than 100 meters.
- The maximum impact was compared to multiple short term health thresholds for the chemical being assessed to determine if a possible acute health risk might exist. These benchmarks are described in the next section of this report.

Two of the source categories (Hydrogen Fluoride and Acetal Resins) in the group of eight did not "screen out" for acute risk; therefore, we performed a more refined assessment. The refined assessment consisted of the following steps:

- Examine aerial photographs of the site to determine if the impact area of concern is outside the facility property boundary.
- Adjust the peak one-hour emissions default (the multiplier of 10) to a more source specific value, where data are available and indicate that such an adjustment is appropriate.
- Perform refined modeling using AERMOD and site specific information.

For source categories with any facilities that still show off-site acute impacts above an HQ of 1 after refining the assessment, we present the maximum HQ values for the available acute thresholds and discuss the possible implications of these results in light of the available health effects information and knowledge regarding the actual facility configuration.

2.6 Dose-Response Assessment

2.6.1 Sources of chronic inhalation dose-response information

Dose-response assessment information (carcinogenic and non-carcinogenic) for chronic exposure for the HAPs reported in the emissions inventory were based on the EPA Office of Air Quality Planning and Standards' existing recommendations for HAPs [*6*], also used for NATA 1999 [*7*]. This information has been obtained from various sources and prioritized according to (1) conceptual consistency with EPA risk assessment guidelines and (2) level of peer review received. The prioritization process was aimed at incorporating into our assessments the best available science with respect to dose-response information. The recommendations are based on the following sources, in order of priority:

1) US Environmental Protection Agency (EPA). EPA has developed dose-response assessments for chronic exposure for many of the pollutants in these risk assessments. These assessments typically provide a qualitative statement regarding the strength of scientific data and specify a reference concentration (RfC, for inhalation) to protect against effects other than cancer and/or a unit risk estimate (URE) to estimate the probability of developing cancer. The RfC is defined as "an estimate (with uncertainty spanning perhaps an order of magnitude) of a continuous inhalation exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime". The URE is defined as "the upper-bound excess cancer risk estimated to result from continuous lifetime exposure to an agent at a concentration of 1 $\mu g/m^3$ in air". EPA disseminates dose-response assessment information in several forms, based on the level of review. The Integrated Risk Information System (IRIS) [*8*] is an EPA database that contains scientific health assessment information, including dose-response information. All IRIS assessments completed since 1996 have also undergone independent external peer review. The current IRIS process includes review by EPA scientists, interagency reviewers from other federal agencies, and the public, and peer review by independent scientists external to EPA. Dose-response assessments for some substances were prepared by the EPA Office of Research and Development, but never submitted for EPA consensus. EPA has assembled the results of many such assessments in the Health Effects Assessment Summary Tables (HEAST)[*9*], which this assessment uses as a source of last resort for one HAP; chloroprene. EPA's science policy approach, under the current carcinogen guidelines, is to use linear low-dose extrapolation as a default option for carcinogens for which the mode of action (MOA) has not been identified. We expect future EPA dose-response assessments to identify nonlinear MOAs where appropriate, and we will use those analyses (once they are peer reviewed) in our risk assessments. At this time, however, there are no available carcinogen dose-response assessments for inhalation exposure that are based on a nonlinear MOA.

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2) US Agency for Toxic Substances and Disease Registry (ATSDR). ATSDR, which is part of the US Department of Health and Human Services, develops and publishes Minimum Risk Levels (MRLs)[*10*] for many toxic substances. The MRL is defined as "an estimate of daily human exposure to a substance that is likely to be without an appreciable risk of

adverse effects (other than cancer) over a specified duration of exposure". ATSDR describes MRLs as substance-specific estimates to be used by health assessors to select environmental contaminants for further evaluation. Exposures above an MRL do not necessarily represent a threat, and MRLs are therefore not intended for use as predictors of adverse health effects or for setting cleanup levels.

3) California Environmental Protection Agency (CalEPA). The CalEPA Office of Environmental Health Hazard Assessment has developed dose-response assessments for many substances, based both on carcinogenicity and health effects other than cancer. The process for developing these assessments is similar to that used by EPA to develop IRIS values and incorporates significant external scientific peer review. As cited in the CalEPA Technical Support Document for developing their chronic assessments[2]: "The guidelines for developing chronic inhalation exposure levels incorporate many recommendations of the U.S. EPA (1994)[3] and NAS (NRC, 1994)[4]." The non-cancer information includes available inhalation health risk guidance values expressed as chronic inhalation reference exposure levels (RELs) [*11*]. CalEPA defines the REL as "the concentration level at or below which no adverse health effects are anticipated in the general human population". CalEPA's quantitative dose-response information on carcinogenicity by inhalation exposure is expressed in terms of the URE [*12*], defined similarly to EPA's URE.

In developing chronic risk estimates, we adjusted dose-response values for some HAPs based on professional judgment, as follows:

1) In the case of the HAP category of glycol ethers, the most conservative dose-response value for any compound in the chemical category (in this case, that is ethylene glycol methyl ether) was used as a surrogate for other compounds in the group for which dose-response values were not available. This was done in order to examine, under conservative assumptions, whether these HAPs that lack dose-response values may pose an unacceptable risk and require further examination, or screen from further assessment.

2) This assessment bases risk estimates for formaldehyde on a dose-response value published in 1999 by the CIIT Centers for Health Research. EPA is currently reviewing the existing IRIS assessment for formaldehyde.

3) For 2 carcinogenic substances, (isophorone and propylene dichloride) that lack inhalation assessments from the sources evaluated in this document, oral carcinogenic potency estimates were converted to inhalation UREs. The

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[2] Air Toxics Hot Spots Program, Risk Assessment Guidelines, Part III - Technical Support Document for the Determination of Noncancer Chronic Reference Exposure Levels. Air Toxicology and Epidemiology Section, Office of Environmental Health Hazard Assessment, California Environmental Protection Agency. February 2000 (http://www.oehha.ca.gov/air/chronic_rels/pdf/relsP32k.pdf)

[3] U.S. EPA. 1994. U.S. Environmental Protection Agency. Methods for Derivation of Inhalation Reference Concentrations and Application of Inhalation Dosimetry. EPA/600/8-90/066F. Office of Research and Development. Washington, DC: U.S.EPA.

[4] NRC. 1994. National Research Council. Science and Judgment in Risk Assessment. Washington, DC: National Academy Press.

conversion from oral risk (per mg/kg/d oral intake) to inhalation risk (per μg/m^3 inhaled) was based on EPA's standard assumptions of a 70-kg body mass and 20 m^3/d inhalation rate, as follows:

$$URE\left(\frac{\mu g}{m^3}\right)^{-1} = CPS\left(\frac{mg}{kg \cdot d}\right)^{-1} \times \frac{1}{70(kg)} \times 20\left(\frac{m^3}{d}\right) \times \frac{1}{1000}\left(\frac{mg}{\mu g}\right)$$

Where: URE = Unit risk estimate for inhalation (risk per μg/m^3)
CPS = Carcinogenic potency slope for ingestion (risk per mg oral intake per kg body mass per day)

EPA understands that conversion of oral dose-response information to inhalation exposure may add significant uncertainty to the resulting risk estimates. However, the alternative to this would have been to omit these substances from quantitative inhalation risk estimates altogether, thereby making a *de facto* assumption of zero carcinogenic potency. For the purposes of the residual risk assessment, EPA prefers to use the approach described above to screen these carcinogens for their potential contributions to risk. If a substance is determined to be a potentially important contributor to risk, it will be prioritized for further dose-response development through EPA's IRIS process.

The emissions inventories for the 8 source categories include emissions of 38 HAP, 14 of which are classified as known, probable, or possible carcinogens, with quantitative cancer dose-response values available. These HAP, the dose-response values used, and the source of the value are listed below in Table 2.6-1.

Table 2.6-1 Dose-Response Values for Chronic Exposure to Carcinogens

URE (unit risk estimate for cancer)[5] = cancer risk per µg/m^3 of average lifetime exposure. Sources: IRIS = EPA Integrated Risk Information System, CAL = California EPA Office of Environmental Health Hazard Assessment, EPA/OAQPS = interim value recommended by the EPA Office of Air Quality Planning and Standards, Conv. Oral = Converted from an oral dose-response assessment.			
Pollutant	**CAS Number**	**URE (1/µg/m3)**	**Source**
Acetaldehyde	75070	2.2E-06	IRIS
Allyl chloride	107051	6.0E-06	CAL
Benzene[6]	71432	7.8E-06	IRIS
Benzyl chloride	100447	4.9E-05	CAL
Bis(2-ethylhexyl)phthalate	117817	2.4E-06	CAL
1,3-Dichloropropene	542756	4.0E-06	IRIS
Epichlorohydrin	106898	1.2E-06	IRIS
Formaldehyde	50000	5.5E-09	EPA/OAQPS
Isophorone	78591	2.7E-07	Conv. Oral[7]
Methylene chloride	75092	4.7E-07	IRIS
Naphthalene	91203	3.4E-05	CAL
Propylene dichloride	78875	1.9E-05	Conv. Oral[8]
Tetrachloroethylene	127184	5.9E-06	CAL
Trichloroethylene	79016	2.0E-06	CAL

The emissions inventories for the 8 source categories include emissions of 35 HAP with quantitative chronic noncancer threshold values available. These HAP, the threshold values used, and the source of the value are listed below in Table 2.6-2.

[5] The URE is the upper-bound excess cancer risk estimated to result from continuous lifetime exposure to an agent at a concentration of 1 µg/m^3 in air. URE's are considered upper bound estimates meaning they represent a plausible upper limit to the true value.

[6] The EPA IRIS assessment for benzene provides a range of plausible UREs. This assessment used the highest value in that range, 7.8E-o6 per µg/m^3 .

[7] No inhalation unit risk estimates were available for this compound, therefore we converted from an IRIS oral potency slope of 0.00095 per mg/kg/d. URE that are converted from the oral route to the inhalation route of exposure are considered highly uncertain, and are only used in cases where no other URE is available.

[8] No inhalation unit risk estimates were available for this compound, therefore we converted from a oral potency slope of 0.068 per mg/kg/d. URE that are converted from the oral route to the inhalation route of exposure are considered highly uncertain, and are only used in cases where no other URE is available.

Table 2.6-2 Dose-Response Values for Chronic Exposure to Noncarcinogens

RfC (reference inhalation concentration) = an estimate (with uncertainty spanning perhaps an order of magnitude) of a continuous inhalation exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime. Sources: IRIS = EPA Integrated Risk Information System, CAL EPA = California EPA Office of Environmental Human Health Assessment, HEAST = EPA Health Effects Assessment Summary Table, ATADR = US Agency for Toxic Substances and Disease Registry.

Pollutant	CAS Number	RfC (mg/m3)	Source[9]
Acetaldehyde	75070	0.009	IRIS -- L
Acetonitrile	75058	0.06	IRIS -- M
Acrolein	107028	0.00002	IRIS -- M
Allyl chloride	107051	0.001	IRIS -- L
Benzene	71432	0.03	IRIS -- M
Bis(2-ethylhexyl)phthalate	117817	0.01	CAL
Chlorine	7782505	0.0002	CAL
Chlorobenzene	108907	1	CAL
Chloroprene	126998	0.007	HEAST
1,3-Dichloropropene	542756	0.02	IRIS -- H
Epichlorohydrin	106898	0.001	IRIS -- M
Ethyl benzene	100414	1	IRIS -- L
Ethyl chloride	75003	10	IRIS -- M
Ethylene glycol	107211	0.4	CAL
Formaldehyde	50000	0.0098	ATSDR
Glycol Ethers		0.02	IRIS -- M
n-Hexane	110543	0.2	IRIS -- M
Hydrochloric acid	7647010	0.02	IRIS -- L
Hydrofluoric acid	7664393	0.014	CAL
Isophorone	78591	2	CAL
Maleic anhydride	108316	0.0007	CAL
Methanol	67561	4	CAL
Methyl chloride	74873	0.09	IRIS -- M
Methyl isobutyl ketone	108101	3	IRIS -- L/M
Methyl methacrylate	80626	0.7	IRIS -- M/H
Methylene chloride	75092	1	ATSDR
Methylene diphenyl diisocyanate	101688	0.0006	IRIS -- M
Naphthalene	91203	0.003	IRIS -- M
Phenol	108952	0.2	CAL
Propylene dichloride	78875	0.004	IRIS -- M
Tetrachloroethylene	127184	0.27	ATSDR
Toluene	108883	0.4	IRIS -- H
Trichloroethylene	79016	0.6	CAL
Vinyl acetate	108054	0.2	IRIS -- H
Xylenes (mixed and o-xylene)	1330207	0.1	IRIS -- M

[9] The descriptors L (low), M (medium), and H (high) have been added for IRIS RfC values to indicate the overall level of confidence in the RfC value, as reported in the IRIS file.

2.6.2 Sources of acute inhalation dose-response information

Hazard identification and dose-response assessment information for acute exposure were based on OAQPS's existing recommendations for HAPs [*13*]. In contrast to the approach for chronic dose-response, no prioritization has been developed for acute noncancer reference values in large part due to the lack of coverage across many chemicals by any one set of reference values specifically designed for this use. We looked to reference values developed for other purposes, such as Reference Exposure Levels (REL), Acute Exposure Guideline Levels (AEGLs), and Emergency Response Planning Guidelines (ERPGs) developed for 1-hour exposure durations.

The California Environmental Protection Agency (CalEPA) has developed acute dose-response assessments for many substances, expressing the results as acute inhalation reference exposure levels, or RELs.

> The acute REL (http://www.oehha.ca.gov/air/pdf/acuterel.pdf) is defined by CalEPA as "the concentration level at or below which no adverse health effects are anticipated for a specified exposure duration[*14*]. RELs are based on the most sensitive, relevant, adverse health effect reported in the medical and toxicological literature. RELs are designed to protect the most sensitive individuals in the population by the inclusion of margins of safety. Since margins of safety are incorporated to address data gaps and uncertainties, exceeding the REL does not automatically indicate an adverse health impact."

The National Advisory Committee for Acute Exposure Guidelines (NAC-AEGL) is a multiple Federal Agency committee that has been responsible for developing Acute Exposure Guideline Levels, or AEGLs. As described in their "Standing Operating Procedures (SOP) of the National Advisory Committee on Acute Exposure Guideline Levels for Hazardous Substances" (http://www.epa.gov/opptintr/aegl/pubs/sop.pdf), "the NRC's previous name for acute exposure levels — community emergency exposure levels (CEELs) — was replaced by the term AEGLs to reflect the broad application of these values to planning, response, and prevention in the community, the workplace, transportation, the military, and the remediation of Superfund sites." This document also states that AEGLs "represent threshold exposure limits for the general public and are applicable to emergency exposures ranging from 10 min to 8 h." The document lays out the purpose and objectives of AEGLs by stating that "the primary purpose of the AEGL program and the NAC/AEGL Committee is to develop guideline levels for once-in-a-lifetime, short-term exposures to airborne concentrations of acutely toxic, high-priority chemicals." In detailing the intended application of AEGL values, the document states that "It is anticipated that the AEGL values will be used for regulatory and nonregulatory purposes by U.S. Federal and State agencies, and possibly the international community in conjunction with chemical emergency response, planning, and prevention programs. More specifically, the AEGL values will be used for conducting various risk assessments to aid in the development of emergency preparedness and prevention

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plans, as well as real-time emergency response actions, for accidental chemical releases at fixed facilities and from transport carriers." The NAC-AEGL defines AEGL-1 and AEGL-2 as:

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> "AEGL-1 is the airborne concentration (expressed as ppm or mg/m^3) of a substance above which it is predicted that the general population, including susceptible individuals, could experience notable discomfort, irritation, or certain asymptomatic nonsensory effects. However, the effects are not disabling and are transient and reversible upon cessation of exposure."

> "AEGL-2 is the airborne concentration (expressed as ppm or mg/m^3) of a substance above which it is predicted that the general population, including susceptible individuals, could experience irreversible or other serious, long-lasting adverse health effects or an impaired ability to escape."

> "Airborne concentrations below AEGL-1 represent exposure levels that can produce mild and progressively increasing but transient and nondisabling odor, taste, and sensory irritation or certain asymptomatic, nonsensory effects. With increasing airborne concentrations above each AEGL, there is a progressive increase in the likelihood of occurrence and the severity of effects described for each corresponding AEGL. Although the AEGL values represent threshold levels for the general public, including susceptible subpopulations, such as infants, children, the elderly, persons with asthma, and those with other illnesses, it is recognized that individuals, subject to unique or idiosyncratic responses, could experience the effects described at concentrations below the corresponding AEGL."

The American Industrial Hygiene Association (AIHA) has developed emergency response planning guidelines (ERPGs) [*15*] for acute exposures at three different levels of severity. These guidelines represent concentrations for exposure of the general population for up to 1 hour associated with effects expected to be mild or transient (ERPG-1), irreversible or serious (ERPG-2), and potentially life-threatening (ERPG-3).

ERPG values (http://www.aiha.org/1documents/Committees/ERP-erpglevels.pdf) are described in their supporting documentation as follows: "Emergency Response Planning Guidelines (ERPGs) were developed for emergency planning and are intended as health based guideline concentrations for single exposures to chemicals. These guidelines (i.e., the ERPG Documents and ERPG values) are intended for use as planning tools for assessing the adequacy of accident prevention and emergency response plans, including transportation emergency planning and for developing community emergency response plans. The emphasis is on ERPGs as planning values: When an actual chemical emergency occurs there is seldom time to measure airborne concentrations and then to take action." ERPG-1 and ERPG-2 values are defined by AIHA as follows:

> "ERPG-1 is the maximum airborne concentration below which it is believed that nearly all individuals could be exposed for up to 1 hour without experiencing other than mild transient adverse health effects or without perceiving a clearly defined, objectionable

odor."

"ERPG-2 is the maximum airborne concentration below which it is believed that nearly all individuals could be exposed for up to 1 hour without experiencing or developing irreversible or other serious health effects or symptoms which could impair an individual's ability to take protective action."

The emissions inventories for the 8 source categories include emissions of 25 HAP with relevant and available quantitative acute dose-response threshold values. These HAP, their acute threshold values used, and the source of the value are listed below in Table 2.6-3.

Table 2.6-3 Dose-Response Values for Acute Exposure

Pollutant	CAS Number	AEGL-1 (1-hr) (mg/m^3)	AEGL-2 (1-hr) (mg/m^3)	ERPG-1 (mg/m^3)	ERPG-2 (mg/m^3)	REL (mg/m^3)
Acetaldehyde	75070			18	360	
Acetonitrile	75058	22	390			
Acrolein	107028	0.069	0.23	0.23	1.1	0.00019
Allyl chloride	107051			9.4	130	
Benzene	71432	170	2600	160	480	1.3
Benzyl chloride	100447			5.2	52	0.24
Chlorine	7782505	1.5	5.8	2.9	8.7	0.21
Epichlorohydrin	106898	19	91	7.6	76	1.3
Ethyl acrylate	140885	34	150	0.041	120	
Formaldehyde	50000	1.1	17	1.2	12	0.094
Glycol Ethers (as ethylene glycol methyl ether)						0.093
Hexane	110543					
Hydrochloric acid	7647010	2.7	33	4.5	30	2.1
Hydrofluoric acid	7664393	0.82	20	1.6	16	0.24
Methanol	67561	690	2700	260	1300	28
Methyl chloride	74873				830	
Methyl methacrylate	80626	70	490			
Methylene chloride	75092			690	2600	14
Methylene diphenyl diisocyanate	101688			0.2	2	
Phenol	108952	58	89	38	190	5.8
Tetrachloroethylene	127184	240	1600	680	1400	20
Toluene	108883	750	1900	190	1100	37
Trichloroethylene	79016	700	2400	540	2700	
Vinyl acetate	108054			18	260	
Xylenes (mixed and o-xylene)	1330207	560	1700			22

2.7 Risk characterization

2.7.1 General

The final product of the risk assessment is the risk characterization, in which the information from the previous steps is integrated and an overall conclusion about risk is synthesized that is complete, informative, and useful for decision makers. In general, the nature of this risk characterization depends on the information available, the application of the risk information and the resources available. In all cases, major issues associated with determining the nature and extent of the risk are identified and discussed. Further, the EPA Administrator's March 1995 *Policy for Risk Characterization* [*16*] specifies that a risk characterization "be prepared in a manner that is clear, transparent, reasonable, and consistent with other risk characterizations of similar scope prepared across programs in the Agency." These principles of transparency and consistency have been reinforced by the *Agency's Risk Characterization Handbook* [*17*], and again in 2002 by the Agency's information quality guidelines [*18*], and in the OMB/OSTP September 2007 Memorandum on Updated Principles for Risk Analysis[10], and are incorporated in these assessments.

Estimates of health risk are presented in the context of uncertainties and limitations in the data and methodology. Through our tiered iterative analytical approach, we have attempted to reduce both uncertainty and bias to the greatest degree possible in these assessments. We have provided summaries of risk metrics for each source category (including maximum individual cancer risks and noncancer hazards, as well as cancer incidence estimates) along with a discussion of the major uncertainties associated with their derivation to provide decision makers with the fullest picture of the assessment and its limitations.

For each carcinogenic HAP included in this assessment that has a potency estimate available, individual and population cancer risks were calculated by multiplying the corresponding lifetime average exposure estimate by the appropriate URE. This calculated cancer risk is defined as the upper-bound probability of developing cancer over a 70-year period (i.e., the assumed human lifespan) at that exposure. Because UREs for most HAPs are upper-bound estimates, actual risks at a given exposure level may be lower than predicted, and could be zero.

Because EPA has not determined that any of the carcinogens listed in Table 2.6-1 have a mutagenic mode of action, [*19*], EPA's *Supplemental Guidance for Assessing Susceptibility from Early-Life Exposure to Carcinogens* [*20*] did not apply to this assessment.

Increased cancer incidence for the entire receptor population within the area of analysis was estimated by multiplying the estimated lifetime cancer risk for each census block by the

[10]Memorandum for the Heads of Executive Departments and Agencies - Updated Principles for Risk Analysis (September 19, 2007), From Susan E. Dudley, Administrator, Office of Information and Regulatory Affairs, Office of Management and Budget; and Sharon L. Hays, Associate Director and Deputy Director for Science, Office of Science and Technology Policy (http://www.whitehouse.gov/omb/memoranda/fy2007/m07-24.pdf)

number of people residing in that block, then summing the results for the entire modeled domain. This lifetime population incidence estimate was divided by 70 years to obtain an estimate of the number of cancer cases per year

In the case of benzene, the high end of the reported cancer URE range was used in our assessment to provide a conservative estimate of potential cancer risks. Use of the high end of the range provides risk estimates that are approximately 3.5 times higher than use of the equally-plausible low end value. Since none of the estimated MIR values exceeded 1 in a million, we did not evaluate the impact of using the low end of the URE range on our risk results.

Unlike linear dose-response assessments for cancer, noncancer health hazards generally are not expressed as a probability of an adverse occurrence. Instead, "risk" for noncancer effects is expressed by comparing an exposure to a reference level as a ratio. The "hazard quotient" (HQ) is the estimated exposure divided by a reference level (e.g., the RfC). For a given HAP, exposures at or below the reference level ($HQ \leq 1$) are not likely to cause adverse health effects. As exposures increase above the reference level (HQs increasingly greater than 1), the potential for adverse effects increases. For exposures predicted to be above the RfC, the risk characterization includes the degree of confidence ascribed to the RfC values for the compound(s) of concern (i.e., high, medium, or low confidence) and discusses the impact of this on possible health interpretations.

In the case of glycol ethers, which can be grouped because of chemical similarity, the most conservative dose-response value of the chemical group available was used as a surrogate for missing dose-response values in the group.

The risk characterization for effects other than cancer is expressed in terms of the HQ for inhalation, calculated for each HAP at each census block centroid. As discussed above, RfCs incorporate generally conservative uncertainty factors in the face of uncertain extrapolations, such that an HQ greater than one does not necessarily suggest the onset of adverse effects. The HQ cannot be translated to a probability that adverse effects will occur, and is unlikely to be proportional to adverse health effect outcomes in a population.

Screening for potentially significant acute inhalation exposures also followed the HQ approach. In this case, we divided the maximum estimated acute exposure by each available short-term threshold value to develop an array of HQ values relative to the various acute endpoints and thresholds. In general, when none of these HQ values are greater than one, there is no potential for acute risk. In those cases where HQ values above one are seen, additional information is needed to determine if there is truly a potential for significant acute risks.

2.7.2 Mixtures

Since most or all receptors in these assessments receive exposures to multiple pollutants rather than a single pollutant, we estimated the aggregate health risks associated with all the exposures from a particular source category combined.

To combine risks across multiple carcinogens, this assessment used the EPA mixtures guidelines' **[*21*,*22*]** default assumption of additivity of effects, and combined risks by summing them using the independence formula in the mixtures guidelines.

In assessing noncancer hazard from chronic exposures, in cases where different pollutants cause adverse health effects via completely different modes of action, it may be inappropriate to aggregate HQs. In consideration of these mode-of-action differences, the mixtures guidelines support aggregating effects of different substances in specific and limited ways. To conform with these guidelines, we aggregated non-cancer HQs of HAPs that act by similar toxic modes of action, or (where this information is absent) that affect the same target organ. This process creates, for each target organ, a target-organ-specific hazard index (TOSHI), defined as the sum of hazard quotients for individual HAPs that affect the same organ or organ system. All TOSHI calculations presented here were based exclusively on effects occurring at the "critical dose" (i.e., the lowest dose that produces adverse health effects). Although HQs associated with some pollutants have been aggregated into more than one TOSHI, this has been done only in cases where the critical dose affects more than one target organ. Because impacts on organs or systems that occur above the critical dose have not been included in the TOSHI calculations, some TOSHIs may have been underestimated. As with the HQ, the TOSHI should not be interpreted as a probability of adverse effects, or as strict delineation of "safe" and "unsafe" levels. Rather, the TOSHI is another measure of the potential for adverse health outcomes associated with pollutant exposure, and needs to be interpreted carefully by health scientists and risk managers.

Because of the conservative nature of the acute inhalation screening and the transient nature of emissions fluctuations and potential exposures, acute impacts were screened on an individual pollutant basis, not using the TOSHI approach.

3 Results Summaries and Risk Characterizations for the Eight Source Categories

In this section, the results of the risk assessments for each of the eight source categories are presented separately. Each subsection includes the following information for each source category:

1) A narrative description of the source category, including a discussion of the processes involved and the number of facilities EPA knows or expects are affected by the MACT standard;

2) A table of emissions for the entire category showing HAP emitted, total source category emission rates for each HAP, and numbers of facilities reporting emissions of each HAP;

3) A table summarizing the chronic inhalation risk results showing the number of facilities modeled, the number of people within 50 km, the MIR for the entire source category, the number of facilities for which the facility-specific MIR exceeds specific cancer and noncancer benchmarks, the number of people for whom the risks exceed the same

benchmarks, the estimated total cancer incidence, and identifying the specific HAPs contributing the most to those risks (HAPs identified as "drivers" include those contributing the most to the risk metric, up to 90% of its value). In addition, this table indicates the maximum HQ from the acute inhalation screening and an indication of how many facilities showed HQ values above 1;

4) In those cases where the acute inhalation screening showed an HQ value greater than 1 for any combination of source and pollutant, a table summarizing the acute screening results showing available acute dose-response values for each affected pollutant, for three effect levels (none, mild, and severe), if available, the maximum acute screening exposure estimated, and the associated HQ values;

5) A narrative summarizing the risk characterization for that category.

Detailed facility-level results for both chronic and acute inhalation risk assessments for each of the eight source categories can be found in Appendix 5.

3.1 Polysulfide Rubber Production

3.1.1 Source Category Description and Results

Polysulfide rubber is a synthetic rubber produced by the reaction of sodium sulfide and p-dichlorobenzene at an elevated temperature in a polar solvent. Specific process steps involved in polysulfide rubber production include preparation of sodium sulfide from aqueous caustic and aqueous sodium hydroxide sulfide in a polar solvent, removal of water from this feedstock by distillation, production of polymer from the sodium sulfide stream and p-dichlorobenzene at elevated temperature in the polar solvent, polymer recovery, washing to remove the sodium chloride produced as a by-product, and drying and packaging. Polysulfide rubber is resilient, resistant to solvents, and has low temperature flexibility, facilitating its use in seals, caulks, automotive parts, rubber molds for casting sculpture, and other products. The primary HAP expected to be emitted by polysulfide rubber production are ethylene oxide, ethylene dichloride, and formaldehyde. The only source of HAP emissions identified for the polysulfide rubber source category in 1995 (at the time the MACT standard was developed) was raw material storage vessels. When EPA developed the MACT rule, one facility was identified for the polysulfide rubber production source category. EPA is aware that this facility has shut down and been dismantled. Therefore, we are unaware of any facilities in this source category that are currently operating in the United States.

The identified facility was located in the draft final 2002 NEI, version 1, and included in the inventory. This facility is classified as a major source in the NEI. The only HAP reported to be emitted from this facility is 4,4'-methylenediphenyl diisocyanate, which is not a PB HAP. The one identified facility was modeled to estimate risk associated with a Polysulfide Rubber production facility if there were one currently in operation.

Tables 3.1-1 and 3.1-2 provide information summarizing emissions and health risks for this source category.

Table 3.1-1 Summary of Emissions from Polysulfide Rubber Production

HAP	Emissions (tpy)	Number of Facilities Reporting HAP (1 facility in Inventory)	Prioritized Chronic Inhalation Dose-Response Value Identified for Use in Screening Risk Assessments by OAQPS		Acute Dose Response values?
			Unit Risk Estimate for Cancer?	Reference Concentration for Noncancer?	
4,4'-Methylenediphenyl Diisocyanate	0.0045	1		✔	✔

Table 3.1-2 Summary of Source Category Level Risks for Polysulfide Rubber

Result		HAP "Drivers"
Facilities in Source Category		
Number of Major Source Facilities Estimated to be in Source Category as of 1995[11]	1	n/a
Number of Facilities Identified in NEI and Modeled in Screening Risk Assessment	1	n/a
Cancer Risks		
Maximum Individual Lifetime Cancer Risk (in 1 million) from any Facility in the Category	--[a]	n/a
Number of Facilities with Maximum Individual Lifetime Cancer Risk:		
Greater than 100 in 1 million	0	n/a
Greater than 10 in 1 million	0	n/a
Greater than 1 in 1 million	0	n/a
Chronic Noncancer Risks		
Maximum Respiratory Hazard Index	<0.01	methylene diphenyl diisocyanate (RfC, medium confidence)
Number of Facilities with Maximum Respiratory Hazard Index:		
Greater than 1	0	n/a
Acute Noncancer Screening Results		
Maximum acute Hazard Quotient	0.0004	methylene diphenyl diisocyanate (ERPG-1)[12]
Number of Facilities With Potential for Acute Effects	0	n/a
Population Exposure		
Number of People Living Within 50 Kilometers of Facilities Modeled	480,000	n/a
Number of People Exposed to Cancer Risk:		
Greater than 100 in 1 million	0	n/a
Greater than 10 in 1 million	0	n/a
Greater than 1 in 1 million	0	n/a
Number of People Exposed to Noncancer Respiratory Hazard Index:		
Greater than 1	0	n/a
Estimated Cancer Incidence (excess cancer cases per year)	--[a]	n/a

Notes:
[a] No facilities in this source category emit a known, probable, or possible human carcinogen.

[11] Hazardous Air Pollutant Emissions from Process Units in the Elastomer Manufacturing Industry—Basis and Purpose Document for Proposed Standards. EPA–453–R–95–006a. U.S. Environmental Protection Agency. May 1995.

[12] Refer to Appendix 5 for HQ values based on ERPG-1 and ERPG-2 thresholds, the only available thresholds for Methylene diphenyl diisocyanate.

3.1.2 Risk Characterization

Current risk levels for this source category are zero since there are no known facilities currently operating in the country. Even when there was one facility operating in the country, the cancer risks and incidence were zero since it did not report emissions of carcinogens, and the maximum chronic noncancer risks corresponded to a respiratory HI less than 0.01, well below 1.0, an exposure level generally considered to be without appreciable health risks. Further, our screening efforts to identify any potential for this source category to pose significant acute inhalation risks, indirect human health risks, or adverse environmental impacts indicated that none of the HAPs emitted by this source category show any such potential.

The major uncertainties associated with the assessment for this source category are those associated with annual emissions of HAP. Several of the HAP expected to be emitted by sources in this category are not included in the inventory. Additionally, default stack parameters were used to estimate the facility impacts. Nonetheless, since there currently are no sources operating in this country, and since the risk results from the refined chronic inhalation assessment and the conservative screening of the only active source in 2002 were so far below levels of concern, we have no information to lead us to believe that future health risks posed by any active sources in this source category (complying with the MACT standard) would be above the level of concern.

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3.2 Ethylene Propylene Rubber Production

3.2.1 Source Category Description and Results

Ethylene propylene elastomer is an elastomer prepared from ethylene and propylene monomers. Ethylene propylene copolymers (EPM) result from the polymerization of the above monomers and contain a saturated chain of the polymethylene type. Ethylene propylene terpolymers (EPDM), also included in this source category, are chemically very similar to EPM but include a third monomer (usually ethylidene norbornene) that is added during the reaction sequence. Common uses for these elastomers include radiator and heater hoses, weather stripping, door and window seals for cars, construction plastics blending, wire and cable insulation and jackets, and single-ply roofing membranes. The most common process for producing EPM and EPDM is a continuous process where the ethylene and propylene monomers are compressed to liquid form and combined in the reactor with the catalyst and solvent. Hexane is the most common solvent used. Sources of HAP emissions for the ethylene propylene elastomer production source category include raw material storage vessels, front-end process vents, back-end process operations, wastewater operations, and equipment leaks. The majority of the emissions come from back-end process operations and equipment leaks. The process "front-end" includes pre-polymerization, reaction, stripping, and material recovery operations; and the process "back-end" includes all operations after stripping (predominately drying and finishing). When EPA developed the MACT rule, four facilities were identified for the ethylene propylene elastomer source category. After the rule was developed, a new ethylene propylene elastomer production facility was constructed that uses a gas phase, fluidized bed process. This new process does not use hexane solvent. The only source of HAP expected from this process is the presence of chlorinated compounds in the catalyst, which resultd in hydrochloric acid emissions when these compounds are sent to the flare.

We found emissions inventory data for all five expected facilities in the 2002 NEI database. For four of the facilities, we replaced the information in the NEI for the identified processes with data collected directly from the industry in 2004. No new data were collected for the fifth and newest facility.

Tables 3.2-1 and 3.2-2 provide information summarizing emissions and health risks for this source category.

Table 3.2-1 Summary of Emissions from Ethylene Propylene Elastomer Production

HAP	Emissions (tpy)	Number of Facilities Reporting HAP (5 facilities in Inventory)	Prioritized Chronic Inhalation Dose-Response Value Identified for Use in Screening Risk Assessments by OAQPS		Acute Dose Response values?
			Unit Risk Estimate for Cancer?	Reference Concentration for Noncancer?	
Hexane	1,023	3		✔	
Hydrochloric Acid	18	1		✔	✔
Methanol	13	1		✔	✔
Toluene	6.8	1		✔	✔
Ethyl Chloride	6.0	1		✔	
Ethylene Glycol	0.0030	1		✔	

Table 3.2-2 Summary of Source Category Level Risks for Ethylene Propylene Elastomer Production

Result		HAP "Drivers"
Facilities in Source Category		
Number of Major Source Facilities Estimated to be in Source Category [13]	5	n/a
Number of Facilities Identified in NEI and Modeled in Screening Risk Assessment	5	n/a
Cancer Risks		
Maximum Individual Lifetime Cancer Risk (in 1 million) from any Facility in the Category	--[a]	n/a
Number of Facilities with Maximum Individual Lifetime Cancer Risk:		
Greater than 100 in 1 million	0	n/a
Greater than 10 in 1 million	0	n/a
Greater than 1 in 1 million	0	n/a
Chronic Noncancer Risks		
Maximum Respiratory Hazard Index	0.5	n-hexane (RfC, medium confidence)
Number of Facilities with Maximum Respiratory Hazard Index:		
Greater than 1	0	n/a
Acute Noncancer Screening Results		
Maximum acute Hazard Quotient	0.3	Toluene (acute REL)[14]
Number of Facilities With Potential for Acute Effects	0	n/a
Population Exposure		
Number of People Living Within 50 Kilometers of Facilities Modeled	1,300,000	n/a
Number of People Exposed to Cancer Risk:		
Greater than 100 in 1 million	0	n/a
Greater than 10 in 1 million	0	n/a
Greater than 1 in 1 million	0	n/a
Number of People Exposed to Noncancer Respiratory Hazard Index:		
Greater than 1	0	n/a
Estimated Cancer Incidence (excess cancer cases per year)	--[a]	n/a

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Notes:
[a] The facilities modeled in this source category do not emit any HAP with a cancer dose-response value. Therefore, there are not any maximum individual lifetime cancer risk results or incidence values.

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[13] Hazardous Air Pollutant Emissions from Process Units in the Elastomer Manufacturing Industry—Basis and Purpose Document for Proposed Standards. EPA–453–R–95–006a. U.S. Environmental Protection Agency. May 1995. *There were 4 facilities in 1995. In addition, EPA is aware that one additional ethylene propylene elastomer facility was constructed following the publication of this document.*

[14] Refer to Appendix 5 for HQ values using all acute thresholds.
[16] Hazardous Air Pollutant Emissions from Process Units in the Elastomer Manufacturing Industry—Basis and Purpose Document for Proposed Standards. EPA–453–R–95–006a. U.S. Environmental Protection Agency. May 1995.

3.2.2 Risk Characterization

There are five facilities identified as being a part of this source category. The results of the risk assessment done showed that the highest noncancer hazard index was 0.5, well below 1.0, an exposure level generally considered to be without appreciable health risks. Further, our screening efforts to identify any potential for this source category to pose significant acute inhalation risks, human health risks from routes of exposure other than inhalation, or adverse environmental impacts indicated that none of the HAPs emitted by this source category show any such potential.

The major uncertainties associated with this category are expected to be the facility-specific information used for modeling (e.g., emissions). For four of the five facilities in this category, we replaced the data that were originally retrieved from the 2002 NEI with site-specific data provided directly from industry for this category. These new data are expected to be less uncertain. Default stack parameters were assigned to a portion of the emission points at the fifth facility, leading to some minor uncertainties in the impacts of that facility. Also, in spite of the nature of the uncertainties ascribed to other aspects of this assessment (e.g., modeling uncertainties) and the nature of any biases on the risk that may result, we believe these results provide a reasonable estimate of the potential maximum and population risks and show this source category to be of low or no risk concern.

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3.3 Butyl Rubber Production

3.3.1 Source Category Description and Results

The Butyl Rubber Production source category includes any facility that manufactures copolymers of isobutylene and isoprene. A typical composition of butyl rubber is approximately 97 percent isobutylene and 3 percent isoprene. Modified, derivative, and halogenated copolymers and latexes are also included in this source category. Butyl rubber is typically made by a precipitation (slurry) polymerization process in which isobutylene and isoprene are copolymerized in methyl chloride solvent. Butyl rubber is very impermeable to common gases and resists oxidation. A specialty group of butyl rubbers are halogenated butyl rubbers, which are produced commercially by dissolving butyl rubber in hydrocarbon solvent and contacting the solution with gaseous or liquid elemental halogens such as chlorine or bromine. Halogenated butyl rubber resists aging to a higher degree than the non-halogenated type and is more compatible with other types of rubber. Uses for butyl rubber include tires, tubes, and tire products; automotive mechanical goods; adhesives, caulks, and sealants; and pharmaceutical uses. Sources of HAP emissions for the butyl rubber source category include front-end process vents (which includes pre-polymerization, reaction, stripping, and material recovery operations), back-end process operations (which includes all operations after stripping, predominately drying and finishing), wastewater operations, and equipment leaks. The majority of the emissions come from front-end process vents and equipment leaks. The primary HAP expected to be emitted from butyl rubber production facilities is methyl chloride. In addition to methyl chloride, hexane, hydrochloric acid, and xylenes are also reported to be emitted from the production of halogenated butyl rubber. We expected two butyl rubber facilities based on work done for the development of the MACT for this source category, and both of these facilities were identified in the 2002 NEI.

Tables 3.3-1 and 3.3-2 provide information summarizing emissions and health risks for this source category.

Table 3.3-1 Summary of Emissions from Butyl Rubber Production

HAP	Emissions (tpy)	Number of Facilities Reporting HAP	Prioritized Chronic Inhalation Dose-Response Value Identified for Use in Screening Risk Assessments by OAQPS		Acute Dose Response values?
			Unit Risk Estimate for Cancer?	Reference Concentration for Noncancer?	
Methyl Chloride	266	2		✔	✔
Hydrochloric Acid	170	2		✔	✔
Hexane	65	1		✔	
Xylenes (Mixture of ortho-, meta-, and para- Isomers)	0.42	1		✔	✔

Table 3.3-2 Summary of Source Category Level Risks for Butyl Rubber Production

Result		HAP "Drivers"
Facilities in Source Category		
Number of Major Source Facilities Estimated to be in Source Category as of 1995[16]	2	n/a
Number of Facilities Identified in NEI and Modeled in Screening Risk Assessment	2	n/a
Cancer Risks		
Maximum Individual Lifetime Cancer Risk (in 1 million) from any Facility in the Category	--[a]	n/a
Number of Facilities with Maximum Individual Lifetime Cancer Risk:		
Greater than 100 in 1 million	0	n/a
Greater than 10 in 1 million	0	n/a
Greater than 1 in 1 million	0	n/a
Chronic Noncancer Risks		
Maximum Neurological Hazard Index	0.2	methyl chloride (RfC, medium confidence)
Number of Facilities with Maximum Neurological Hazard Index:		
Greater than 1	0	n/a
Acute Noncancer Screening Results		
Maximum Acute Hazard Quotient	0.1	methyl chloride (ERPG-2)[17]
Number of Facilities With Potential for Acute Effects	0	n/a
Population Exposure		
Number of People Living Within 50 Kilometers of Facilities Modeled	3,500,000	n/a
Number of People Exposed to Cancer Risk:		
Greater than 100 in 1 million	0	n/a
Greater than 10 in 1 million	0	n/a
Greater than 1 in 1 million	0	n/a
Number of People Exposed to Noncancer Neurological Hazard Index:		
Greater than 1	0	n/a
Estimated Cancer Incidence (excess cancer cases per year)	--[a]	n/a

Notes:
[a] The facilities modeled in this source category do not emit any known, probable, or possible human carcinogens.

[17] The ERPG-2 threshold is the only available acute threshold for methyl chloride.

3.3.2 Risk Characterization

There are only two facilities identified as being a part of this source category. The results of the risk assessment indicated that there are no cancer risks since no carcinogens were reported as emitted and that the highest noncancer hazard index was 0.1, well below 1, the exposure level generally considered to be without appreciable health risks. Further, our screening efforts to identify any potential for this source category to pose significant acute inhalation risks, human health risks from routes of exposure other than inhalation, or adverse environmental impacts indicated that none of the HAPs emitted by this source category show any such potential.

The major uncertainties associated with this category are those associated with the facility-specific information used for modeling (e.g., emissions). We revised the data that were originally retrieved from the 2002 NEI with site-specific data provided directly from industry representatives from sources in this category in 2004. These new data are considered to be quite accurate. Also, in spite of the nature of the uncertainties ascribed to other aspects of this assessment (e.g., modeling uncertainties) and the nature of any biases on the risk that may result, we believe these results provide a reasonable estimate of the potential maximum risks as well as the distribution of risks across the population.

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3.4 Neoprene Production

3.4.1 Source Category Description and Results

Neoprene is a polymer of chloroprene. The polymer structure can be modified by copolymerizing chloroprene (e.g., with sulfur or 2,3-dichloro-1,3-butadiene) to produce a range of materials with varying chemical and physical properties. Neoprene was originally developed as an oil-resistant substitute for natural rubber, and its properties allow its use in a wide variety of applications including wetsuits, gaskets and seals, hoses and tubing, plumbing fixtures, adhesives, and other products. Sources of HAP emissions for the neoprene production source category include raw material storage vessels, front-end process vents, back-end process operations, wastewater operations, and equipment leaks. Most of the emissions come from front-end process vents. The process "front-end" includes pre-polymerization, reaction, stripping, and material recovery operations; and the process "back-end" includes all operations after stripping (predominately drying and finishing). The primary HAP emitted by production are hydrogen chloride and chloroprene, and toluene. When EPA developed the MACT rule, three facilities were identified for the neoprene production source category. After reviewing the 2002 NEI we found only one facility known to still be in operation. We believe this to be the only major source currently operating. For this one facility in the source category, we replaced the information in the NEI for the neoprene processes with data collected directly from industry in 2004. Chloroprene and toluene are the only two HAP reported as emissions from this facility.

Tables 3.4-1 and 3.4-2 provide information summarizing emissions and health risks for this source category.

Table 3.4-1 Summary of Emissions from Neoprene Production

HAP	Emissions (tpy)	Number of Facilities Reporting HAP (1 facility in Inventory)	Prioritized Chronic Inhalation Dose-Response Value Identified for Use in Screening Risk Assessments by OAQPS		Acute Dose Response values?
			Unit Risk Estimate for Cancer?	Reference Concentration for Noncancer?	
Chloroprene	232	1		✔	
Toluene	57	1		✔	✔

Table 3.4-2 Summary of Source Category Risks for Neoprene Production

Result		HAP "Drivers"
Facilities in Source Category		
Number of Major Source Facilities Estimated to be in Source Category as of 1995[18]	3	n/a
Number of Facilities Identified in NEI and Modeled in Screening Risk Assessment	1	n/a
Cancer Risks		
Maximum Individual Lifetime Cancer Risk (in 1 million) from any Facility in the Category	--[a]	n/a
Number of Facilities with Maximum Individual Lifetime Cancer Risk:		
Greater than 100 in 1 million	0	n/a
Greater than 10 in 1 million	0	n/a
Greater than 1 in 1 million	0	n/a
Chronic Noncancer Risks		
Maximum Respiratory Hazard Index	0.8	Chloroprene (HEAST)
Number of Facilities with Maximum Respiratory Hazard Index:		
Greater than 1	0	n/a
Acute Noncancer Screening Results		
Maximum Acute Hazard Quotient	0.4	Toluene (acute REL)[19]
Number of Facilities With Potential for Acute Effects	0	n/a
Population Exposure		
Number of People Living Within 50 Kilometers of Facilities Modeled	1,000,000	n/a
Number of People Exposed to Cancer Risk:		
Greater than 100 in 1 million	0	n/a
Greater than 10 in 1 million	0	n/a
Greater than 1 in 1 million	0	n/a
Number of People Exposed to Noncancer Respiratory Hazard Index:		
Greater than 1	0	n/a
Estimated Cancer Incidence (excess cancer cases per year)	--[a]	n/a

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Notes:
[a] The facility modeled in this source category do not emit HAP that are known, probable, or possible human carcinogens.

[18] Hazardous Air Pollutant Emissions from Process Units in the Elastomer Manufacturing Industry—Basis and Purpose Document for Proposed Standards. EPA–453–R–95–006a. U.S. Environmental Protection Agency. May 1995.
[19] Refer to Appendix 5 for acute HQ values using all acute thresholds.
[21] Refer to Appendix 5 for HQ values using all acute thresholds.

3.4.2 Risk Characterization

Only one facility in this source category is currently believed to be operating. Cancer risks associated with the facility are zero since there are no emissions of known, probable, or possible human carcinogens. The maximum noncancer hazard index was 0.8, indicating an exposure level considered to be without appreciable risk of adverse health impacts. Our screening efforts to identify any potential for this source category to pose significant human health risks from routes of exposure other than inhalation or adverse environmental impacts indicated that no such potential exists.

The major uncertainties associated with this category are those associated with the facility-specific information used for modeling (e.g., emissions). We revised the data that were originally retrieved from the 2002 NEI with data provided directly from industry representatives for the one known facility in this category. These new data are expected to be quite accurate. Because many of the processes are batch but the emissions data are expressed as annual totals, there is some uncertainty associated with the acute risk screening, yet since no exceedances of acute thresholds were found using the conservative assumption that peak emissions exceed annual average emissions by a factor of 10, we believe no potential exists for adverse health effects from acute exposures. Overall, in spite of the nature of the uncertainties described for other aspects of this assessment (e.g., modeling uncertainties) and the nature of any biases on the risk that may result, we believe these results provide a reasonable estimate of the potential maximum and population risks and show this source category to be of low or no risk concern.

3.5 Epoxy Resins Production

3.5.1 Source Category Description and Results

The epoxy resins production source category includes operations located at major sources that manufacture basic liquid epoxy resins (BLR). These resins are plastic materials that become hard, infusible solids upon the addition of a hardening agent. They are used in the production of glues, adhesives, plastic parts, and surface coatings. BLR do not include specialty epoxy resins or modified epoxy resins (i.e., BLR that have been blended with solvents, reactive diluents, or other resins).

The methods used for BLR production include both batch and continuous operations. BLR is manufactured from epichlorohydrin (1-chloro-2,3-epoxypropane) and bisphenol-A in either a conventional process or two-step process. When the MACT standard for this category was developed in 1994, U.S. production of BLR was primarily carried out via the two-step process, in which epichlorohydrin and bisphenol-A are coupled followed by a dehydrochlorination of the product into BLR. Processes resulting in HAP emissions include epichlorohydrin charging and reaction in the reactor for the initiation of BLR production and removal of epichlorohydrin from the final product either before or after dehydrochlorination. HAP air emissions from these processes can be released as breathing and working losses from storage tanks, products vented from process vessels, leaks from piping equipment used to transfer HAP compounds (equipment leaks), and volatilized HAP from wastewater streams. The primary HAP expected to be emitted from the BLR production process is epichlorohydrin. As of 1994, three U.S. facilities were known to be producing BLR. All three of these facilities were major sources and thus subject to the MACT rule for epoxy resins production. We found the three facilities in the 2002 NEI database and replaced the data with new more recent data obtained from industry. We are not aware of any new facilities that have begun operation since 1994.

Tables 3.5-1 and 3.5-1 provide information summarizing emissions and health risks for this source category.

Table 3.5-1 Summary of Emissions from Epoxy Resins Production

HAP	Emissions (tpy)	Number of Facilities Reporting HAP (3 facilities in Inventory)	Prioritized Chronic Inhalation Dose-Response Value Identified for Use in Screening Risk Assessments by OAQPS		Acute Dose Response values?
			Unit Risk Estimate for Cancer?	Reference Concentration for Noncancer?	
1-Chloro-2,3-Epoxypropane (Epichlorohydrin)	11.8	3	✔	✔	✔
Chlorobenzene	1.6	2		✔	
Phenol	0.48	1		✔	✔
Xylenes (Mixture of o, m, and p Isomers)	0.48	1		✔	✔
Ethyl benzene	0.48	2		✔	
o-Xylene	0.47	1			✔
Propylene dichloride	0.08	2	✔	✔	✔
Allyl chloride	0.04	2	✔	✔	✔
1,3-Dichloropropene	0.01	2	✔	✔	
Glycol ethers	0.01	1		✔	✔
Methyl chloride	0.01	1		✔	✔
Toluene	0.005	1		✔	✔
Acrolein	0.003	1		✔	✔
Benzyl Chloride	0.002	1	✔		✔
Ethyl acrylate	0.0000005	1			✔

Table 3.5-2 Summary of Source Category Level Risks for Epoxy Resins Production

Result		HAP "Drivers"
Facilities in Source Category		
Number of Facilities Estimated to be in the source category in 1994 (from 4/29/04 Fact Sheet Accompanying the Proposed Rule)	3	n/a
Number of Facilities Modeled in Screening Risk Assessment	3	n/a
Cancer Risks		
Maximum Individual Lifetime Cancer Risk (in 1 million) from any Facility in the Category	0.1	epichlorohydrin
Number of Facilities with Maximum Individual Lifetime Cancer Risk:		
Greater than 100 in 1 million	0	n/a
Greater than 10 in 1 million	0	n/a
Greater than 1 in 1 million	0	n/a
Chronic Noncancer Risks		
Maximum Respiratory Hazard Index	0.08	epichlorohydrin (RfC, medium confidence)
Number of Facilities with Maximum Respiratory Hazard Index:		
Greater than 1	0	n/a
Acute Noncancer Screening Results		
Maximum Acute Hazard Quotient	0.6	epichlorohydrin (acute REL)[21]
Number of Facilities With Potential for Acute Effects	0	
Population Exposure		
Number of People Living Within 50 Kilometers of Facilities Modeled	3,700,000	n/a
Number of People Exposed to Cancer Risk:		
Greater than 100 in 1 million	0	n/a
Greater than 10 in 1 million	0	n/a
Greater than 1 in 1 million	0	n/a
Number of People Exposed to Noncancer Respiratory Hazard Index:		
Greater than 1	0	n/a
Estimated Cancer Incidence (excess cancer cases per year)	0.00002	epichlorohydrin
Contribution of HAP to Cancer Incidence		
epichlorohydrin	73%	n/a
propylene dichloride	18%	n/a
allyl chloride	3%	n/a
1,3-dichloropropene	2%	n/a

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3.5.2 Risk Characterization

There are three facilities identified as being a part of this source category, and they were all included in the assessment. The results of the risk assessment showed the highest individual cancer risk to be 0.1-in-1 million, significantly below 1-in-1 million. The noncancer hazard index was 0.1, well below 1.0, an exposure level generally considered to be without appreciable health risks. Further, our screening efforts to identify any potential for this source category to pose significant acute inhalation risks, indirect human health risks, or adverse environmental impacts indicate that none of the HAPs emitted by this source category show any such potential.

The major uncertainties associated with this category are those associated with the facility-specific information used for modeling (e.g., emissions). Since we revised the data that was originally retrieved from the 2002 NEI with data provided directly from industry representatives for this category, we believe the uncertainties associated with them are quite small. Also, in spite of the nature of the uncertainties described for other aspects of this assessment (e.g., modeling uncertainties) and the nature of any biases on the risk that may result, we believe these results provide a reasonable estimate of the potential maximum and population risks and shows this source category to be of low or no risk concern.

3.6 Non-nylon Polyamides Production

3.6.1 Source Category Description and Results

The non-nylon polyamides production source category includes sources that manufacture epichlorohydrin cross-linked non-nylon polyamides. These products include polyamide resins made with dibasic esters, dicarboxylic acids, amines, and epichlorohydrin. The resins are used primarily by the paper industry as an additive to increase the tensile strength of paper products. Natural polymers, such as those contained in paper products, have little crosslinking, which allows their fibers to change position or separate completely when in contact with water. The addition of epichlorohydrin cross-linked non-nylon polyamides to these polymers causes the formation of a stable polymeric web among the natural fibers. Because the polymeric web holds the fibers in place even in the presence of water, epichlorohydrin cross-linked non-nylon polyamides are also referred to as wet-strength resins (WSR).

WSR production can be achieved via both batch and continuous operations, but most manufacturing occurs in single batch reactors. The process begins with the transfer of feedstock prepolymers, epichlorohydrin, and other reactive chemicals from storage tanks to weighing tanks. The reactor is charged with prepolymers and the other reactive chemicals to initiate the reaction. Epichlorohydrin is then added to induce the cross-linking reaction. When sufficient crosslinking has been achieved, an acid is added to the reactor to halt the reaction, and water, excess acid, and excess feedstocks are removed from the reactor. Emissions associated with the batch reactor are produced during reactor charging, reaction heat-up, and acid additions. Epichlorohydrin can potentially be emitted during each of these events, and hydrochloric acid can be emitted if it is used to halt the crosslinking reaction. Emissions of methanol are also possible, depending on what chemicals are reacted with the prepolymers. HAP air emissions from these processes can be released as breathing and working losses from storage tanks, displacement emissions from weigh tanks, products vented from process vessels, leaks from piping equipment used to transfer HAP compounds (equipment leaks), and, to a small extent, volatilized HAPs from wastewater streams. When the non-nylon polyamides production MACT rule was proposed in 1994, there were at least 17 facilities manufacturing WSR nationwide. Of these facilities, nine were considered major sources and thus subject to the rule. Recent EPA investigation has determined that there are four plants currently subject to the rule, and all four of these were included in our assessment. The other five sources either closed, merged, or are no longer major sources.

Tables 3.6-1 and 3.6-2 provide information summarizing emissions and health risks for this source category.

Table 3.6-1 Summary of Emissions from the Non-Nylon Polyamides Production Source Category

HAP	Emissions (tpy)	Number of Facilities Reporting HAP (4 facilities in Inventory)	Prioritized Chronic Inhalation Dose-Response Value Identified for Use in Screening Risk Assessments by OAQPS		Acute Dose Response values?
			Unit Risk Estimate for Cancer?	Reference Concentration for Noncancer?	
1-Chloro-2,3-Epoxypropane (Epichlorohydrin)	4.1	4	✔	✔	✔
Hydrochloric acid	2.3	2		✔	✔

Table 3.6-2 Summary of Source Category Level Risks for Non-Nylon Polyamides Production

Result		**HAP "Drivers"**
Facilities in Source Category		
Number of Facilities Estimated to be in the source category in 1994 (from 4/29/04 Fact Sheet Accompanying the Proposed Rule)	17[23]	n/a
Number of Facilities Modeled in Screening Risk Assessment	4	n/a
Cancer Risks		
Maximum Individual Lifetime Cancer Risk (in 1 million) from any Facility in the Category	0.4	epichlorohydrin
Number of Facilities with Maximum Individual Lifetime Cancer Risk:		
Greater than 100 in 1 million	0	n/a
Greater than 10 in 1 million	0	n/a
Greater than 1 in 1 million	0	n/a
Chronic Noncancer Risks		
Maximum Respiratory Hazard Index	0.3	epichlorohydrin (RfC, medium confidence)
Number of Facilities with Maximum Respiratory Hazard Index:		
Greater than 1	0	n/a
Acute Noncancer Screening Results		
Maximum Acute Hazard Quotient	0.02	epichlorohydrin (acute REL)[24]
Number of Facilities With Potential for Acute Effects	0	n/a
Population Exposure		
Number of People Living Within 50 Kilometers of Facilities Modeled	2,300,000	n/a
Number of People Exposed to Cancer Risk:		
Greater than 100 in 1 million	0	n/a
Greater than 10 in 1 million	0	n/a
Greater than 1 in 1 million	0	n/a
Number of People Exposed to Noncancer Respiratory Hazard Index:		
Greater than 1	0	n/a
Estimated Cancer Incidence (excess cancer cases per year)	0.00003	epichlorohydrin
Contribution of HAP to Cancer Incidence		
epichlorohydrin	100%	n/a

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[23] EPA estimates that there are currently four sources subject to the MACT rule.
[24] Refer to Appendix 5 for HQ values using all acute thresholds.
[26] Refer to Appendix 5 for HQ values using all acute thresholds.

3.6.2 Risk Characterization

Current risk levels for this source category are low, with a maximum individual lifetime cancer risk of 0.4 in a million and estimated cancer incidence of 0.00003 cases per year. The maximum chronic noncancer HI (respiratory) is 0.3, well below the value of 1 that is the exposure level generally considered to be without appreciable health risks. Further, our screening efforts to identify any potential for this source category to pose significant acute inhalation risks, indirect human health risks, or adverse environmental impacts indicate that none of the HAPs emitted by this source category show any such potential.

The major uncertainties associated with this category are those associated with the facility-specific information used for modeling (e.g., emissions). Since we revised the data that was originally retrieved from the 2002 NEI with data provided directly from industry representatives for this category, we believe the uncertainties associated with them are quite small. Many processes in this source category are batch, so emissions are not constant. However, the uncertainty associated with short-term emissions estimates is mitigated by the fact that our conservative acute screening assessment indicated no potential for exceeding any acute thresholds. Also, in spite of the nature of the uncertainties described for other aspects of this assessment (e.g., modeling uncertainties) and the nature of any biases on the risk that may result, we believe these results provide a reasonable estimate of the potential maximum and population risks and shows this source category to be of low or no risk concern.

3.7 Hydrogen Fluoride Production

3.7.1 Source Category Description and Results

The HF source category includes any facility engaged in the production and recovery of HF by reacting calcium fluoride with sulfuric acid. This category does not include any process that produces gaseous HF for direct reaction with hydrated aluminum to form aluminum fluoride. (In these processes, HF is not recovered as an intermediate or final product prior to reacting with the hydrated aluminum.) Potential sources of HAP emissions at HF production facilities include: process vents on HF recovery and refining equipment; storage vessels used to store HF; bulk loading of tank trucks and tank rail cars; leaks from HF handling equipment; and reaction kiln seal leaks. The only HAP expected to be emitted from the processes in this source category is HF.

At the time of MACT development in 1998, production of HF in the U.S. occurred at two facilities: an area source located in Louisiana and a major source located in Texas. However, we believe that both facilities are currently operating as major sources and included both in our assessment. A third facility, located in Kentucky, has been shut down, and was not included in our assessment; however production at that facility could resume in the future.

Tables 3.7-1 and 3.7-2 provide information summarizing emissions and health risks for this source category.

Table 3.7-1 Summary of Emissions from the Hydrogen Fluoride Source Category

HAP	Emissions (tpy)	Number of Facilities Reporting HAP (2 facilities in Inventory)	Prioritized Chronic Inhalation Dose-Response Value Identified for Use in Screening Risk Assessments by OAQPS		Acute Dose Response values?
			Unit Risk Estimate for Cancer?	Reference Concentration for Noncancer?	
Hydrogen Fluoride	8	2		✔	✔

Table 3.7-2 Summary of Source Category Level Risks for Hydrogen Fluoride

Result		HAP "Drivers"
Facilities in Source Category		
Number of Facilities in Source Category in 1998, from the MACT Proposal Preamble (63 FR 55181 October 14, 1998	2	n/a
Number of Facilities Identified in NEI and Modeled in Screening Risk Assessment	2	n/a
Cancer Risks		
Maximum Individual Lifetime Cancer Risk (in 1 million) from any Facility in the Category	--[a]	n/a
Number of Facilities with Maximum Individual Lifetime Cancer Risk:		
Greater than 100 in 1 million	0	n/a
Greater than 10 in 1 million	0	n/a
Greater than 1 in 1 million	0	n/a
Chronic Noncancer Risks		
Maximum Skeletal Hazard Index	<0.01	hydrofluoric acid (chronic REL)
Number of Facilities with Maximum Respiratory Hazard Index:		
Greater than 1	0	n/a
Acute Noncancer Screening Results		
Maximum Acute Hazard Quotient	20, 6.7	hydrofluoric acid (REL, AEGL-1)[26]
Number of Facilities With Potential for Acute Effects	2	n/a
Acute Noncancer Refined Results		
Maximum Acute Hazard Quotient	0.2, 0.06	hydrofluoric acid (REL, AEGL-1)
Number of Facilities With Potential for Acute Effects	0	n/a
Population Exposure		
Number of People Living Within 50 Kilometers of Facilities Modeled	3,500,000	n/a
Number of People Exposed to Cancer Risk:		
Greater than 100 in 1 million	0	n/a
Greater than 10 in 1 million	0	n/a
Greater than 1 in 1 million	0	n/a
Number of People Exposed to Noncancer Respiratory Hazard Index:		
Greater than 1	0	n/a
Total Cancer Incidence (excess cancer cases per year)	--[a]	n/a

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Notes:
[a] The facilities modeled in this source category do not emit any HAP with a cancer dose-response value. Therefore, there are not any maximum individual lifetime cancer risk results or incidence values.

3.7.2 Risk Characterization

Since no known carcinogens are emitted from the HF source category, cancer risks were not estimated. The maximum chronic noncancer hazard index is predicted to be less than 0.01 for respiratory effects, significantly less than 1, the exposure level generally considered to be without appreciable health risk. Our screening efforts to identify any potential for this source category to pose significant indirect human health risks or adverse environmental impacts indicated that no such potential exists.

There are two identified operating facilities for the HF source category. The assessment for acute impacts showed that the two HF facilities did not "screen out" and that for one facility both the "mild effect" level (AEGL-1) and "no effects" level (California REL) for hydrofluoric acid could be exceeded under worst-case meteorological conditions if maximum hourly emissions of hydrofluoric acid exceed their average hourly emission rate by a factor of 10. Since both facilities in the HF production source category did not screen-out for potential acute impacts, we performed additional site-specific assessments (see Appendix 6, Figures 1-4). We contacted the permitting agency and a process engineer at one of the facilities to gather additional source specific information. Based on discussions with the permitting agency and the process engineer, we determined that these facilities operate continuously and that the peak emissions are not expected to exceed twice the hourly average. By adjusting the short-term emission rate to more accurately represent the true facility operating conditions (from 10 to 2), no offsite impacts above the REL (HQ_{REL} = 0.34) or AEGL-1 (HQ_{AEGL-1} = 0.1) were predicted from the first facility. For the second facility that exceeded both the REL and AEGL-1, we remodeled using the AERMOD model to more accurately predict the worst case acute impacts. By adjusting the short-term emission rate to more accurately represent facility operating conditions (from 10 to 2), no exceedances of the AEGL-1 (HQ_{AEGL-1} = 0.06) and REL (HQ_{REL} = 0.2) were predicted to occur outside the facility property boundary.

The major uncertainties associated with the assessment for this source category are those associated with annual emissions of HAP. While default stack parameters were used to estimate the impacts for approximately 30% of the emission points at the 2 facilities assessed, we consider this to be a minor source of uncertainty in the assessment. The HAPs included in the inventory are those that are expected from this source category, and the two facilities assessed are believed to be the only two facilities currently in operation. In addition, we are aware that some concerns have been expressed regarding the adequacy of our screening for potential adverse environmental effects for the pollutant hydrogen fluoride. Indeed, there is a significant lack of scientific understanding and assessment methodologies for such potential adverse environmental effects. Notwithstanding these concerns, we believe that the negative outcomes of our assessments for acute and chronic noncancer human health endpoints provides strong support for our conclusion that adverse environmental impacts are not expected for hydrogen fluoride emissions from this source category.

3.8 Acetal Resins Production

3.8.1 Source Category Description and Results

The acetal resins production source category includes any operation that manufactures homopolymers or copolymers of alternating oxymethylene units. Acetal resins, also known as polyoxymethylenes, polyacetals, or aldehyde resins, are a type of plastic possessing relatively high strength and rigidity without being brittle. They have good frictional properties and are resistant to moisture, heat, fatigue, and solvents. These qualities facilitate the use of acetal resins as parts in a variety of industrial applications, including gears, bearings, bushings, and various other moving parts in appliances and machines. Acetal resins are also used in a range of consumer products, such as automotive door handles, seat belt components, plumbing fixtures, shaver cartridges, zippers, and gas tank caps. Acetal resins are generally produced by polymerizing formaldehyde (HCHO) with the methylene functional group (CH_2), and they are characterized by repeating oxymethylene units (CH_20) in the polymer backbone. In 1998, when the acetal resin MACT rule was proposed, there were three facilities in the U.S. producing acetal resins. Using information collected by the MACT engineers, three acetal resin production facilities were identified in the 2002 NEI. Individual records at these were assigned process MACT codes based on the Source Classification Code (SCC) and pollutant emission records.

Emission sources from acetal resin production include storage vessels that hold process feed materials, process vents, process wastewater treatment systems, and equipment leaks from compressors, agitators, pressure relief devices, sampling connection systems, valves, connectors, and instrumentation systems. The storage vessels associated with acetal resin production are primarily used for storage of solvents, and HAP emissions released from the storage vessels are typically organic HAPs. Front end process vents are associated with formaldehyde emissions. Back end process vent emissions occur from reactor units, mixing vessels, solvent recovery operations, and other operations and can emit ethylene oxide. Wastewater streams from acetal resin plants contain formaldehyde and methanol. Overall, the primary HAPs expected to be emitted during acetal resin production are formaldehyde and methanol.

Tables 3.8-1 and 3.8-2 provide information summarizing emissions and health risks for this source category.

Table 3.8-1 Summary of Emissions from Acetal Resins Production

HAP	Emissions (tpy)	Number of Facilities Reporting HAP (3 facilities in Inventory)	Prioritized Chronic Inhalation Dose-Response Value Identified for Use in Screening Risk Assessments by OAQPS		Acute Dose Response values?
			Unit Risk Estimate for Cancer?	Reference Concentration for Noncancer?	
Methanol	126	3		✔	✔
Formaldehyde	61	3	✔	✔	✔
Chlorine	10	1		✔	✔
Toluene	2	2		✔	✔
Allyl Chloride	2	1	✔	✔	✔
Benzene	1	1	✔	✔	✔
Hexane	0.9	3		✔	
Methyl Chloride	0.7	1		✔	✔
Naphthalene	0.2	1	✔	✔	
Xylenes (Mixture of o, m, and p Isomers)	0.2	1		✔	✔
Epichlorohydrin (1-Chloro-2,3-Epoxypropane)	0.1	1	✔	✔	✔
Acetaldehyde	0.08	1	✔	✔	✔
Phenol	0.03	2		✔	✔
Methyl Isobutyl Ketone	0.07	1		✔	
Acetonitrile	0.01	2		✔	✔
Isophorone	0.01	1	✔	✔	

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Table 3.8-2 Summary of Source Category Level Risks for Acetal Resins Production

Result		HAP "Drivers"
Facilities in Source Category		
Number of Facilities Estimated to be Subject to MACT in Source Category in 1998, from the Proposal Preamble (63 FR 55178, October 14 8, 1998)	3	n/a
Number of Facilities Modeled in Screening Risk Assessment	3	n/a
Cancer Risks		
Maximum Individual Lifetime Cancer Risk (in 1 million) from any Facility in the Category	0.3	allyl chloride
Number of Facilities with Maximum Individual Lifetime Cancer Risk:		
Greater than 100 in 1 million	0	n/a
Greater than 10 in 1 million	0	n/a
Greater than 1 in 1 million	0	n/a
Chronic Noncancer Risks		
Maximum Respiratory Hazard Index	0.2	chlorine (chronic REL)

Result		HAP “Drivers”
Number of Facilities with Maximum Respiratory Hazard Index:		
Greater than 1	0	n/a
Acute Noncancer Screening Results		
Maximum Acute Hazard Quotient	50, 4	formaldehyde (REL, AEGL-1)[28]
Number of Facilities With Potential for Acute Effects	3	n/a
Acute Noncancer Refined Results		
Maximum Acute Hazard Quotient	1.7, 0.14	formaldehyde (REL, AEGL-1)[29]
Number of Facilities With Potential for Acute Effects	1	n/a
Population Exposure		
Number of People Living Within 50 Kilometers of Facilities Modeled	1,200,000	n/a
Number of People Exposed to Cancer Risk:		
Greater than 100 in 1 million	0	n/a
Greater than 10 in 1 million	0	n/a
Greater than 1 in 1 million	0	n/a
Number of People Exposed to Noncancer Respiratory Hazard Index:		
Greater than 1	0	n/a
Estimated Cancer Incidence (excess cancer cases per year)	0.00004	allyl chloride
Contribution of HAP to Cancer Incidence		
allyl chloride	52%	n/a
benzene	6%	n/a
napthalene	40%	n/a
Bis(2-ethylhexyl)phthalate	2%	n/a

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3.8.2 Risk Characterization

Current risk levels for the acetal resins source category are below the level of concern for cancer and chronic noncancer. The maximum individual lifetime cancer risk from any facility in the category is 0.3 in 1 million and the maximum chronic noncancer target- organ specific hazard index (TOSHI) is 0.1. Both levels are generally considered to be without appreciable health risks. The total estimated cancer incidence from these facilities is 0.00004 excess cancer cases per year. Further, our screening efforts to identify any potential for this source category to pose significant indirect human health risks or adverse environmental impacts indicated that none of the HAPs emitted by this source category show any such potential.

The screening assessment for acute impacts suggests that short-term formaldehyde concentrations at the three modeled facilities could exceed acute thresholds, assuming worst-case meteorological conditions are present, using our default assumption that the maximum hourly emissions of formaldehyde exceed the average hourly emission rate by a factor of ten.

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[28] Refer to Appendix 5 for HQ values using all acute thresholds.

[29] The acute REL for formaldehyde is based upon a mild eye irritation toxicity endpoint [14]. The AEGL-1 for formaldehyde is based upon a moderate eye irritation toxicity endpoint [24].

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One of the facilities showed potential exceedances of the California REL level only, and two facilities showed potential exceedances of both the REL level and the AEGL-1 level. Examination of the initial screening results for the first facility showed that the potential exceedances did not extend off the facility site. We performed additional site specific assessments of the other two facilities (see Appendix 6, Figures 5-10). Discussions with a plant engineer for one facility revealed that the acetal resins processes operate continuously and that a reasonable worst-case emissions multiplier would be 1.5 instead of our default 10 multiplier. We performed more refined modeling (AERMOD) for these two facilities using the emissions multiplier of 1.5. The results for the second facility indicated no potential for exceeding the AEGL -1 (HQ = 0.14) and showed that potential exceedances of the REL do not extend off-site, except for a small extension over a river to the north of the facility. The maximum off-site HQ_{REL} corresponding to this location is 1.7 over the river. The analysis showed that meteorological conditions resulting in exceedances of the REL (i.e., HQ_{REL} values above 1) may occur up to only about 0.02 percent of the time or about 2 hours per year along the river. Thus we believe, the actual potential for adverse acute health effects surrounding this facility is low. The results for the third facility showed no potential for exceeding the AEGL-1 (HQ_{AEGL-1} = 0.14), and that the potential for exceeding the REL extends off-site to the south along a roadway. The maximum off-site HQ_{REL} for this facility is 1.6. The analysis showed that meteorological conditions resulting in exceedances of the REL (i.e., HQ_{REL} values above 1) may occur up to 46 hours per year along the roadway. Additionally, the third facility reports that current actual emissions for this facility are significantly less than those used for this assessment, since one of the higher emission sources listed for this facility in the 2002 NEI data has been shut down and is no longer in operation. Figure 10 in Appendix 6 depicts the AERMOD prediction of what the acute risk levels are expected to be today, based on the lower current emissions. Figure 10 shows that all potential impacts occur within the facility property boundary.

The major uncertainties associated with the risk assessment for this source category are those associated with annual emissions of HAP. One facility reports emissions of benzene and allyl chloride, which are two relatively toxic HAP not expected to be emitted from this source category. Since the risk assessment shows allyl chloride to be the cancer risk driver for the source category, this indicates a potential overestimate in the cancer estimate. The complete source category is covered by our assessment and a majority of the stack parameters used to estimate risk impacts were site-specific values, not defaults, providing only a minor source of uncertainty. Finally, it should be noted that there are HAP for which dose-response values are currently under review (particularly formaldehyde and methanol) for cancer, chronic non-cancer, or acute effects. For these HAP, an understatement/overstatement of risk is possible if revised assessments determine that these HAP are more/less potent than currently thought.

4 General Discussion of Uncertainties and How They Have Been Addressed

While each of the source-category-specific sections in the preceding section discusses specific uncertainties unique to each category, the following general discussion of uncertainties applies to all of them.

4.1 Exposure Modeling Uncertainties

Emission inventory uncertainties have been previously discussed for each source category. In general, each of these source categories currently includes only a few sources, and to our knowledge, all of the sources in each category were included in our assessment. Further, the emissions levels have been reviewed by experts familiar with the processes and deemed to be generally consistent with their knowledge.

The chronic exposure modeling uncertainties are considered relatively small since we are using EPA's refined local dispersion model with site-specific parameters and reasonably representative meteorology. If anything, the population exposure estimates are biased high by not accounting for short- or long-term population mobility, and by neglecting processes like deposition, plume depletion, and atmospheric degradation. Additionally, estimates of the maximum individual risk (MIR) contain uncertainty, because they are derived at census block centroid locations rather than actual residences. This uncertainty is known to create potential underestimates and overestimates of the actual MIR values for individual facilities, but, overall, it is not thought to have a significant impact on the estimated MIR for a source category. Finally, we did not factor in the possibility of a source closure occurring during the 70-year chronic exposure period, leading to a potential upward bias in both the MIR and population risk estimates; nor did we factor in the possibility of population growth during the 70-year chronic exposure period, leading to a potential downward bias in both the MIR and population risk estimates.

As previously discussed in section 2.2, a sensitivity analysis performed for the 1999 NATA found that the selection of the meteorology dataset location could result in a range of chronic ambient concentrations which varied from as much as 17% below the predicted value to as much as 84% higher than the predicted value. This variability translates directly to the predicted exposures and risks in our assessment, indicating that the actual risks could vary from 17% lower to 84% higher than the predicted values.

We have purposely biased the acute screening results high, considering that they depend upon the joint occurrence of independent factors, such as hourly emissions rates, meteorology and human activity patterns. Furthermore, in cases where multiple acute threshold values are considered scientifically acceptable we have chosen the most conservative of these assessments, erring on the side of overestimating potential health risks from acute exposures. In the cases where these results indicated the potential for exceeding short-term health thresholds, we have refined our assessment by developing a better understanding of the geography of the facility relative to potential exposure locations and the true variability of short-term emission rates. In each of these cases, we have determined that this refined information reduced the likelihood of acute health concerns.

4.2 Uncertainties in the Dose-Response Relationships

In the sections that follow, separate discussions are provided on uncertainty associated with cancer potency factors and for noncancer reference values. Cancer potency values are derived for chronic (lifetime) exposures. Noncancer reference values are generally derived for chronic exposures (up to a lifetime), but may also be derived for acute (<24 hours), short-term

(>24 hours up to 30 days), and subchronic (>30 days up to 10% of lifetime) exposure durations, all of which are derived based on an assumption of continuous exposure throughout the duration specified. For the purposes of assessing all potential health risks associated with the emissions included in this assessment, we rely on both chronic (cancer and noncancer) and acute (noncancer) benchmarks, which are described in more detail below.

Although every effort is made to identify peer-reviewed dose-response values for all 188 HAPs emitted by the sources included in this assessment, some HAP have no peer-reviewed cancer potency values or reference values for chronic non-cancer or acute effects. Since exposures to these pollutants cannot be included in a quantitative risk estimate, an understatement of risk for these pollutants at environmental exposure levels is possible.

Additionally, chronic dose-response values for 12 of the compounds included in this assessment are currently under EPA IRIS review and revised assessments may determine that these pollutants are more or less potent than currently thought. We will re-evaluate residual risks if, as a result of these reviews, a dose-response metric changes enough to indicate that the risk assessment supporting today's notice may significantly mischaracterize human health risk.

Cancer assessment

The discussion of dose-response uncertainties in the estimation of cancer risk below focuses on the uncertainties associated with the specific approach currently used by the EPA to develop cancer potency factors. In general, these same uncertainties attend the development of cancer potency factors by CalEPA, the source of peer-reviewed cancer potency factors used where EPA-developed values are not yet available. To place this discussion in context, we provide a quote from the EPA's *Guidelines for Carcinogen Risk Assessment*.[*23*] "The primary goal of EPA actions is protection of human health; accordingly, as an Agency policy, risk assessment procedures, including default options that are used in the absence of scientific data to the contrary, should be health protective." The approach adopted in this document is consistent with this approach as described in the *Cancer Guidelines*.

For cancer endpoints EPA usually derives an oral slope factor for ingestion and a unit risk value for inhalation exposures. These values allow estimation of a lifetime probability of developing cancer given long-term exposures to the pollutant. Depending on the pollutant being evaluated, EPA relies on both animal bioassay and epidemiological studies to characterize cancer risk. As a science policy approach, consistent with the *Cancer Guidelines*, EPA uses animal cancer bioassays as indicators of potential human health risk when other human cancer risk data are unavailable.

Extrapolation of study data to estimate potential risks to human populations is based upon EPA's assessment of the scientific database for a pollutant using EPA's guidance documents and other peer-reviewed methodologies. The EPA *Guidelines for Carcinogen Risk Assessment* describes the Agency's recommendations for methodologies for cancer risk assessment. EPA believes that cancer risk estimates developed following the procedures described in the *Cancer Guidelines* and outlined below generally provide an upper bound estimate of risk. That is, EPA's upper bound estimates represent a "plausible upper limit to the true value of a quantity" (although this is usually not a true statistical confidence limit).[30] In some circumstances, the true risk could be as low as zero; however, in other circumstances the risk could also be greater.[31] When developing an upper bound estimate of risk and to provide risk values that do not underestimate risk, EPA generally relies on conservative default approaches.[32] EPA also uses the upper bound (rather than lower bound or central) estimates in its assessments, although it is noted that this approach can have limitations for some uses (e.g. priority setting, expected benefits analysis).

Such health risk assessments have associated uncertainties, some which may be considered quantitatively, and others which generally are expressed qualitatively. Uncertainties may vary substantially among cancer risk assessments associated with exposures to different pollutants, since the assessments employ different databases with different strengths and limitations and the procedures employed may differ in how well they represent actual biological processes for the assessed substance. EPA's *Risk Characterization Handbook* also recommends that risk characterizations present estimates demonstrating the impact on the assessment of alternative choices, data, models and assumptions (U.S. EPA, 2000). Some of the major sources of uncertainty and variability in deriving cancer risk values are described more fully below.

(1) The qualitative similarities or differences between tumor responses observed in experimental animal bioassays and those which would occur in humans is a source of uncertainty in cancer risk assessment. In general, EPA does not assume that tumor sites observed in an experimental animal bioassay are necessarily predictive of the sites at which tumors would occur in humans.[33] However, unless scientific support is available to show otherwise, EPA assumes that tumors in animals are relevant in humans, regardless of target organ concordance. For a specific pollutant, qualitative differences in species responses can lead to either under-estimation or over-estimation of human cancer risks.

(2) Uncertainties regarding the most appropriate dose metric for an assessment can also lead to differences in risk predictions. For example, the measure of dose is commonly expressed in units of mg/kg/d ingested or the inhaled concentration of the pollutant. However, data may support development of a pharmacokinetic model for the absorption,

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[30] IRIS glossary (www.epa.gov/NCEA/iris/help_gloss.htm).

[31] The exception to this is the URE for benzene, which is considered to cover a range of values, each end of which is considered to be equally plausible, and which is based on maximum likelihood estimates.

[32] According to the NRC report *Science and Judgment in Risk Assessment* (NRC, 1994) "[Default] options are generic approaches, based on general scientific knowledge and policy judgment, that are applied to various elements of the risk-assessment process when the correct scientific model is unknown or uncertain." The 1983 NRC report *Risk Assessment in the Federal Government: Managing the Process* defined *default option* as "the option chosen on the basis of risk assessment policy that appears to be the best choice in the absence of data to the contrary" (NRC, 1983a, p. 63). Therefore, default options are not rules that bind the agency; rather, the agency may depart from them in evaluating the risks posed by a specific substance when it believes this to be appropriate. In keeping with EPA's goal of protecting public health and the environment, default assumptions are used to ensure that risk to chemicals is not underestimated (although defaults are not intended to overtly overestimate risk). See EPA 2004 *An Examination of EPA Risk Assessment Principles and Practices,* EPA/100/B-04/001 available at: http://www.epa.gov/osa/pdfs/ratf-final.pdf.

[33] Per the EPA Cancer Guidelines: "The default option is that positive effects in animal cancer studies indicate that the agent under study can have carcinogenic potential in humans." and "Target organ concordance is not a prerequisite for evaluating the implications of animal study results for humans."

distribution, metabolism and excretion of an agent, which may result in improved dose metrics (e.g., average blood concentration of the pollutant or the quantity of agent metabolized in the body). Quantitative uncertainties result when the appropriate choice of a dose metric is uncertain or when dose metric estimates are themselves uncertain (e.g., as can occur when alternative pharmacokinetic models are available for a compound). Uncertainty in dose estimates may lead to either over or underestimation of risk.

(3) For the quantitative extrapolation of cancer risk estimates from experimental animals to humans, EPA uses scaling methodologies (relating expected response to differences in physical size of the species), which introduce another source of uncertainty. These methodologies are based on both biological data on differences in rates of process according to species size and empirical comparisons of toxicity between experimental animals and humans. For a particular pollutant, the quantitative difference in cancer potency between experimental animals and humans may be either greater than or less than that estimated by baseline scientific scaling predictions due to uncertainties associated with limitations in the test data and the correctness of scaled estimates.

(4) EPA cancer risk estimates, whether based on epidemiological or experimental animal data, are generally developed using a benchmark dose (BMD) analysis to estimate a dose at which there is a specified excess risk of cancer (a POD). Statistical uncertainty in developing a POD using a benchmark dose (BMD) approach is generally addressed though use of the 95% lower confidence limit on the dose at which the specified excess risk occurs (the BMDL), decreasing the likelihood of understating risk. EPA has generally utilized the multistage model for estimation of the BMDL using cancer bioassay data (see further discussion below).

(5) Extrapolation from high to low doses is an important, and potentially large, source of uncertainty in cancer risk assessment. EPA uses different approaches to low dose risk assessment (i.e., developing estimates of risk for exposures to environmental doses of an agent from observations in experimental or epidemiological studies at higher dose) depending on the available data and understanding of a pollutant's mode of action (i.e., the manner in which a pollutant causes cancer). EPA's cancer guidelines express a preference for the use of reliable, compound-specific, biologically-based risk models when feasible; however, such models are rarely available. The mode of action for a pollutant (i.e., the manner in which a pollutant causes cancer) is a key consideration in determining how risks should be estimated for low-dose exposure. A reference value is calculated when the available mode of action data show the response to be nonlinear (e.g., as in a threshold response). A linear low-dose (straight line from POD) approach is used when available mode of action data support a linear (e.g., nonthreshold response) or as the most common default approach when a compound's mode of action is unknown. Linear extrapolation can be supported by both pollutant-specific data and broader scientific considerations. For example, EPA's *Cancer Guidelines* generally consider a linear dose-response to be appropriate for pollutants that interact with DNA and induce mutations. Pollutants whose effects are additive to background biological processes in cancer development can also be predicted to have low-dose linear responses, although the slope of this relationship may not be the same as the slope estimated by the straight line approach.

EPA most frequently utilizes a linear low-dose extrapolation approach as a baseline science-policy choice (a "default") when available data do not allow a compound-specific determination. This approach is designed to not underestimate risk in the face of uncertainty and variability. EPA believes that linear dose-response models, when appropriately applied as part of EPA's cancer risk assessment process, provide an upper bound estimate of risk and generally provide a health protective approach. Note that another source of uncertainty is the characterization of low-dose nonlinear, non-threshold relationships. The National Academy of Sciences has encouraged the exploration of sigmoidal type functions (e.g., log-probit models) in representing dose response relationships due to the variability in response within human populations. A recent National Research Council report (NRC, 2006)[34] suggests that models based on distributions of individual thresholds are likely to lead to sigmoidal-shaped dose-response functions for a population. This report notes sources of variability in the human population: "One might expect these individual tolerances to vary extensively in humans depending on genetics, coincident exposures, nutritional status, and various other susceptibility factors..." Thus, if a distribution of thresholds approach is considered for a carcinogen risk assessment, application would depend on ability of modeling to reflect the degree of variability in response in human populations (as opposed to responses in bioassays with genetically more uniform rodents). Note also that low dose linearity in risk can arise for reasons separate from population variability: due to the nature of a mode of action and additivity of a chemical's effect on top of background chemical exposures and biological processes.

As noted above, EPA's current approach to cancer risk assessment typically utilizes a straight line approach from the BMDL. This is equivalent to using an upper confidence limit on the slope of the straight line extrapolation. The impact of the choice of the BMDL on bottom line risk estimates can be quantified by comparing risk estimates using the BMDL value to central estimate BMD values, although these differences are generally not a large contributor to uncertainty in risk assessment (Subramaniam et al., 2006). It is important to note that earlier EPA assessments, including the majority of those for which risk values exist today, were generally developed using the multistage model to extrapolate down to environmental dose levels and did not involve the use of a POD. Subramaniam et al (2006) also provide comparisons indicating that slopes based on straight line extrapolation from a POD do not show large differences from those based on the upper confidence limit of the multistage model.

(6) Cancer risk estimates do not generally make specific adjustments to reflect the variability in response within the human population — resulting in another source of uncertainty in assessments. In the diverse human population, some individuals are likely to be more sensitive to the action of a carcinogen than the typical individual, although compound-specific data to evaluate this variability are generally not available. There may also be important life stage differences in the quantitative potency of carcinogens and, with the exception of the recommendations in EPA's *Supplemental Cancer Guidance* for carcinogens with a mutagenic mode of action, risk assessments do not generally quantitatively address life stage differences. However, one approach used commonly in EPA assessments

[34] NRC (National Research Council) 2006. Assessing the Human Health Risks of Trichloroethylene. National Academies Press, Washington DC

that may help address variability in response is to extrapolate human response from results observed in the most sensitive species and sex tested, resulting typically in the highest URE which can be supported by reliable data, thus supporting estimates that are designed not to underestimate risk in the face of uncertainty and variability.

Chronic noncancer assessment

Chronic noncancer reference values represent chronic exposure levels that are intended to be health-protective. That is, EPA and other organizations which develop noncancer reference values (e.g., the Agency for Toxic Substances and Disease Registry – ATSDR) utilize an approach that is intended not to underestimate risk in the face of uncertainty and variability. When there are gaps in the available information, uncertainty factors (UFs) are applied to derive reference values that are intended to be protective against appreciable risk of deleterious effects. Uncertainty factors are commonly default values[35] e.g., factors of 10 or 3, used in the absence of compound-specific data; where data are available, uncertainty factors may also be developed using compound-specific information. When data are limited, more assumptions are needed and more default factors are used. Thus there may be a greater tendency to overestimate risk—in the sense that further study might support development of reference values that are higher (i.e., less potent) because fewer default assumptions are needed. However, for some pollutants it is possible that risks may be underestimated.

For non-cancer endpoints related to chronic exposures, EPA derives a Reference Dose (RfD) for exposures via ingestion, and a Reference Concentration (RfC) for inhalation exposures. These values provide an estimate (with uncertainty spanning perhaps an order of magnitude) of daily oral exposure (RfD) or of a continuous inhalation exposure (RfC) to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime.[36] To derive values that are intended to be "without appreciable risk," EPA's methodology relies upon an uncertainty factor (UF) approach (U.S. EPA, 1993, 1994) which includes consideration of both uncertainty and variability.

EPA begins by evaluating all of the available peer-reviewed literature to determine non-cancer endpoints of concern, evaluating the quality, strengths and limitations of the available studies. EPA typically chooses the relevant endpoint that occurs at the lowest dose, often using statistical modeling of the available data, and then determines the appropriate point of departure (POD) for derivation of the reference value. A POD is determined by (in order of

[35] According to the NRC report *Science and Judgment in Risk Assessment* (NRC, 1994) "[Default] options are generic approaches, based on general scientific knowledge and policy judgment, that are applied to various elements of the risk-assessment process when the correct scientific model is unknown or uncertain." The 1983 NRC report *Risk Assessment in the Federal Government: Managing the Process* defined *default option* as "the option chosen on the basis of risk assessment policy that appears to be the best choice in the absence of data to the contrary" (NRC, 1983a, p. 63). Therefore, default options are not rules that bind the agency; rather, the agency may depart from them in evaluating the risks posed by a specific substance when it believes this to be appropriate. In keeping with EPA's goal of protecting public health and the environment, default assumptions are used to ensure that risk to chemicals is not underestimated (although defaults are not intended to overtly overestimate risk). See EPA 2004 *An examination of EPA Risk Assessment Principles and Practices,* EPA/100/B-04/001 available at: http://www.epa.gov/osa/pdfs/ratf-final.pdf.

[36] See IRIS glossary

preference): (1) a statistical estimation using the benchmark dose (BMD) approach; (2) use of the dose or concentration at which the toxic response was not significantly elevated (no observed adverse effect level— NOAEL); or (3) use of the lowest observed adverse effect level (LOAEL).

A series of downward adjustments using default UFs is then applied to the POD to estimate the reference value (U.S. EPA 1994, 2002). While collectively termed "UFs", these factors account for a number of different quantitative considerations when utilizing observed animal (usually rodent) or human toxicity data in a risk assessment. The UFs are intended to account for: (1) variation in susceptibility among the members of the human population (i.e., inter-individual variability); (2) uncertainty in extrapolating from experimental animal data to humans (i.e., interspecies differences); (3) uncertainty in extrapolating from data obtained in a study with less-than-lifetime exposure (i.e., extrapolating from subchronic to chronic exposure); (4) uncertainty in extrapolating from a LOAEL in the absence of a NOAEL; and (5) uncertainty when the database is incomplete or there are problems with applicability of available studies. When scientifically sound, peer-reviewed assessment-specific data are not available, default adjustment values are selected for the individual UFs. For each type of uncertainty (when relevant to the assessment), EPA typically applies an UF value of 10 or 3 with the cumulative UF value leading to a downward adjustment of 10-3000 fold from the selected POD. An UF of 3 is used when the data do not support the use of a 10-fold factor. If an extrapolation step or adjustment is not relevant to an assessment (e.g., if applying human toxicity data and an interspecies extrapolation is not required) the associated UF is not used. The major adjustment steps are described more fully below.

1) Heterogeneity among humans is a key source of variability as well as uncertainty. Uncertainty related to human variation is considered in extrapolating doses from a subset or smaller-sized population, often of one sex or of a narrow range of life stages (typical of occupational epidemiologic studies), to a larger, more diverse population. In the absence of pollutant-specific data on human variation, a 10-fold UF is used to account for uncertainty associated with human variation. Human variation may be larger or smaller; however, data to examine the potential magnitude of human variability are often unavailable. In some situations, a smaller UF of 3 may be applied to reflect a known lack of significant variability among humans.

2) Extrapolation from results of studies in experimental animals to humans is a necessary step for the majority of chemical risk assessments. When interpreting animal data, the concentration at the POD (e.g. NOAEL, BMDL) in an animal model (e.g. rodents) is extrapolated to estimate the human response. While there is long-standing scientific support for the use of animal studies as indicators of potential toxicity to humans, there are uncertainties in such extrapolations. In the absence of data to the contrary, the typical approach is to use the most relevant endpoint from the most sensitive species and the most sensitive sex in assessing risks to the average human. Typically, compound specific data to evaluate relative sensitivity in humans versus rodents are lacking, thus leading to uncertainty in this extrapolation. Size-related differences (allometric relationships) indicate that typically humans are more sensitive than rodents when compared on a mg/kg/day basis. The default choice of 10 for the interspecies UF is consistent with these differences. For a specific chemical, differences in species responses may be greater or less than this value.

Pharmacokinetic models are useful to examine species differences in pharmacokinetic processing and associated uncertainties; however, such dosimetric adjustments are not always possible. Information may not be available to quantitatively assess toxicokinetic or toxicodynamic differences between animals and humans, and in many cases a 10-fold UF (with separate factors of 3 for toxicokinetic and toxicodynamic components) is used to account for expected species differences and associated uncertainty in extrapolating from laboratory animals to humans in the derivation of a reference value. If information on one or the other of these components is available and accounted for in the cross-species extrapolation, a UF of 3 may be used for the remaining component.

3) In the case of reference values for chronic exposures where only data from shorter durations are available (e.g., 90-day subchronic studies in rodents) or when such data are judged more appropriate for development of an RfC, an additional UF of 3 or 10-fold is typically applied unless the available scientific information supports use of a different value.

4) Toxicity data are typically limited as to the dose or exposure levels that have been tested in individual studies; in an animal study, for example, treatment groups may differ in exposure by up to an order of magnitude. The preferred approach to arrive at a POD is to use BMD analysis; however, this approach requires adequate quantitative results for a meaningful analysis, which is not always possible. Use of a NOAEL is the next preferred approach after BMD analysis in determining a POD for deriving a health effect reference value. However, many studies lack a dose or exposure level at which an adverse effect is not observed (i.e., a NOAEL is not identified). When using data limited to a LOAEL, a UF of 10 or 3-fold is often applied.

5) The database UF is intended to account for the potential for deriving an underprotective RfD/RfC due to a data gap preventing complete characterization of the chemical's toxicity. In the absence of studies for a known or suspected endpoint of concern, a UF of 10 or 3-fold is typically applied.

Acute noncancer assessment

Many of the UFs used to account for variability and uncertainty in the development of acute reference values are quite similar to those developed for chronic durations, but more often using individual UF values that may be less than 10. UFs are applied based on chemical-specific or health effect-specific information (e.g., simple irritation effects do not vary appreciably between human individuals, hence a value of 3 is typically used), or based on the purpose for the reference value (see the following paragraph). The UFs applied in acute reference value derivation include: 1) heterogeneity among humans; 2) uncertainty in extrapolating from animals to humans; 3) uncertainty in LOAEL to NOAEL adjustments; and 4) uncertainty in accounting for an incomplete database on toxic effects of potential concern. Additional adjustments are often applied to account for uncertainty in extrapolation from observations at one exposure duration (e.g., 4 hours) to arrive at a POD for derivation of an acute reference value at another exposure duration (e.g., 1 hour).

Not all acute reference values are developed for the same purpose and care must be taken when interpreting the results of an acute assessment of human health effects relative to the reference value or values being exceeded. Where relevant to the estimated exposures, the lack of threshold values at different levels of severity should be factored into the risk characterization as potential uncertainties.

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